Risk Assessment Methodologies: EPA's Responses to Questions from the National Academy of Sciences

## Table of Contents

## LIST OF FIGURES

## LIST OF TABLES

## APPENDICES

| APPENDIX | |
|---|---|
| A | Developmental Toxicity Guidelines |
| B | Cancer Risks from Outdoor Exposure to Air Toxics |
| C | Examples of Past Hazardous Air Pollutant Decisions |
| D | Addition of Benzene to List of HAPs |
| E | Health Risk and Economic Impact Assessments of Suspected Carcinogens |
| F | Carcinogen Assessments Group's Final Report on Population Risk to Ambient Benzene Exposures |
| G | Proposed Airborne Carcinogen Policy |
| H | Integrated Simulation Model for Health Risk Assessment |
| I | Proposed Emission Standards for Maleic Anhydride Plants |
| J | Withdrawal of Proposed Standards |
| K | Transmittal of Interim Quantitative Cancer Unit Risk Estimates Due to Inhalation of Benzene |
| L | Vinyl Chloride Opinion |
| M | Briefing on Benzene for the EPA Administrator |
| N | Proposed Benzene NESHAP Decisions - Memorandum by the EPA Administrator |
| O | Proposed Benzene NESHAP |

## APPENDIX

| | |
|---|---|
| P | Briefing for the Assistant Administrator Summarizing Public Comments Following Benzene Proposal |
| Q | Promulgation of Standards for Several Benzene Source Categories |
| R | Benzene Questions and Answers |
| S | Advance Briefing for News Media on Benzene Risk Assessment |
| T | Tiered Modeling Approach for Assessing Risk |
| U | Monte Carlo Approach |
| V | Geographic Information System - Application to Exposure Modeling |
| W | Hazardous Air Pollutant Exposure Model - Mobility Considerations |

**Note: The appendices contain full and abbreviated versions of larger documents. A complete copy of all the abbreviated references has been provided to the NAS staff.**

Question 1: What does EPA consider to be the risk assessment requirements needed to implement the Clean Air Act of 1990?

## I.A Introduction

Implementation of Title III of the Clean Air Act (CAA) requires the development and consideration of risk and hazard assessment in several provisions. The extent of assessment appropriate for each implementation activity is dependent on various factors. These include, but are not limited to, the purpose of the specific provision, the statutory timing and relationship to other provisions, and the availability of data and analytical methods. The next sections describe the regulatory flow and timing of Title III implementation, identify the levels of assessment and review, and describe the provisions with risk-related requirements.

## I.B Regulatory Flow and Chronology of Title III Implementation

Regulation under Title III is comprised of two major steps: the application of technology-based emission standards to categories of major stationary industrial sources, followed by the evaluation of residual risks and the development of further standards, as necessary, to insure that public health is being protected with an ample margin of safety. Affected source categories are identified based on emissions of listed pollutants. The list of source categories and agenda for regulation are required to be published. Extensions from compliance with the technology-based standards are available with demonstration of voluntary emissions reductions, documented problems with the installation of controls, or recently installed controls. Following compliance with the technology-based standards (maximum achievable control technology or MACT), EPA is required to evaluate residual risks and promulgate further standards, if necessary. Compliance and enforcement of the regulations is implemented through an operating permit program at the State level. The flow of the regulatory program under Title III is summarized in Figure 1.

In addition to the regulatory requirements, there are a number of studies in Title III that require reports to Congress on various schedules. The timing of these studies and the principal regulatory milestones are illustrated in Figure 2.

## I.C Levels of Risk Assessment

Table 1 presents a brief overview of those Title III provisions which contain elements of risk assessment. Included is a categorization of the level of analysis associated with each activity and the level of review. These are briefly described below. Their use, as exemplified in the past and present or future efforts is presented in the response to Question 2.

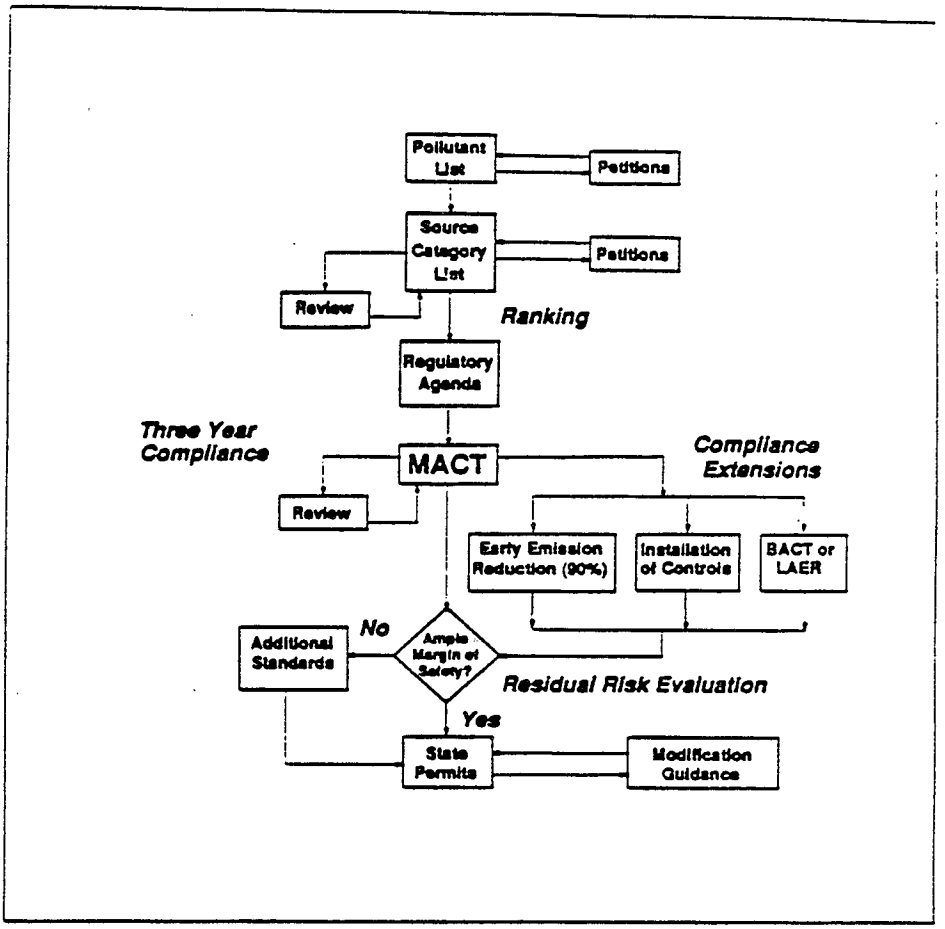


Figure 1: Title III Regulatory Flow

a. Problem Definition: Problem definition activities generally include scoping studies to broadly assess the potential magnitude of the air toxics problem.

b. Hazard Assessment: A hazard assessment is the evaluation of the potential of a substance to cause human health or environmental effects. It would include an assessment of the available effects data and additional information such as environmental fate, potential for bioaccumulation, and identification of sensitive subpopulations.

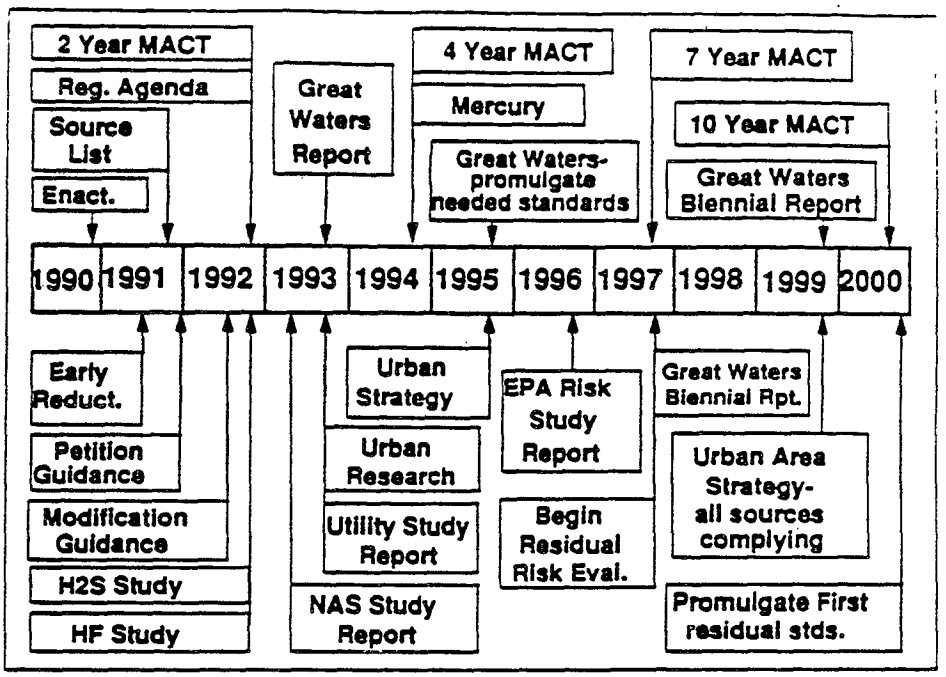


Figure 2: Chronology of Title III Risk-Related Activities

c. Hazard Ranking: A hazard ranking is the relative comparison of information identified in individual pollutant hazard assessments. The purpose of this type of analysis is to rank or group pollutants that pose similar hazards to public health or the environment.

d. Risk Ranking: A risk ranking is the comparative ranking that considers both emissions or exposure information and health effects data. The data may vary in quality depending upon the needs of the specific project.

e. Quantitative Risk Assessment: Quantitative risk assessment is the quantitative characterization of individual and population risk. It is typically conducted for individual sources, but the results may also be aggregated across an industrial source category. This level of analysis requires the most extensive collection of data and analytical resources.

## I.D Risk Assessment Review Requirements

The assessments and methods used to implement various aspects of the air

toxics program undergo a series of internal and external review procedures. The level of the review varies but will generally fall into one or more of the categories. The levels of review intended for each implementation activity under Title III are indicated in Table 1 and are broadly described below. It should be noted that individual components of a risk assessment may have a formal peer review. For example, hazard assessment documents always undergo external peer review.

a. Internal Review: This generally consists of review by EPA technical and scientific staff, supervisors, and senior management. It may also include review by Agency-wide committees such as the Risk Assessment Forum (RAF) or the Risk Assessment Council (RAC). Internal review is included in all phases of regulatory and methods development.

b. External Review by Individuals: This review is conducted by individuals outside the Agency who are selected for their expertise in a specific area.

c. External Review by Panels: Such review is the result of a workshop or meeting of experts and representatives of interested groups or affected organizations.

d. Public Review: This consists of review by the public of all supporting documentation as part of the formal rulemaking process, and follows publication of a proposed rule in the Federal Register.

e. Formal External Review: This is review by established advisory committees (e.g., EPA's Science Advisory Board (SAB), National Academy of Sciences (NAS), National Air Pollution Control Techniques Advisory Committee (NAPCTAC)).[1]

## I.E Title III Risk-Related Provisions

Several provisions of Title III contain requirements for risk or hazard assessment. Beginning on the following page, Table 1 summarizes these provisions. The levels of analysis and review identified on the Table correspond to the levels discussed above. The codes used in the Table are explained in notes on the last page of the Table.

---

[1]The NAPCTAC is a committee composed of representatives of industry, environmental groups, and State and local agencies. It was established pursuant to Section 117 of the CAA. The primary focus of NAPCTAC is the review of control technology alternatives considered in the development of emission standards. The role has expanded to include other areas of relevant to Title III implementation.

Table 1: Overview of CAA Title III Risk-Related Requirements

| Statutory Cite/Project Description | Statutory Deadline | Data Considered for Finding | Level of Analysis[1] | Level of Review[2] |
|---|---|---|---|---|
| Section 112(a) Lesser Quantity Emission Rates<br><br>- modification of major source definition based on potency, persistence, bioaccumulation potential, other characteristics of a pollutant, or other relevant factors<br>- EPA currently considering implementation options | - discretionary activity | - sufficient data to identify critical health effect(s)<br>- estimate of risk based on generic exposure modeling<br>- consideration of bioaccumulation | RR | a,c,d |
| Section 112(b) Pollutant Listing and Delisting<br><br>- modification of hazardous air pollutant (HAP) list by EPA on own accord or following petition<br>- listing of HAP requires demonstration that emissions, ambient concentrations, bioaccumulation, or deposition of substance is known or reasonably anticipated to cause an adverse human health or environmental effect<br>- delisting of HAP requires opposite demonstration<br>- EPA currently drafting guidance for petitioners | - periodic review of list<br><br>- response to petition within 18 months of receipt | - identification of known or reasonably anticipated adverse effect(s)<br>Hazard data: sufficient to identify critical effect(s)<br>Exposure data: sufficient to identify potential exposures | RR | a,d,e |

Table 1: Overview of CAA Title III Risk-Related Requirements

| Statutory Cite/Project Description | Statutory Deadline | Data Considered for Finding | Level of Analysis[1] | Level of Review[2] |
|---|---|---|---|---|
| Section 112(c) Source Category List (Delisting)<br><br>- modification of source category list by EPA on own accord or following petition<br>- max. individual cancer risk from any 1 source cannot >$10^{-6}$<br>- noncancer risks: must protect public with "ample margin of safety"<br>- consideration of environmental effects included<br>- EPA currently drafting guidance for petitioners | - development of list within 12 months; list proposed 6/91<br><br>- response to petition within 12 months of receipt | - identification of effects and exposure as listed above<br>- potential exposures may be assessed using tiered approach with increasing data requirements | QRA | a,d |
| Section 112(c) Source Category List (Schedule)<br><br>- identification of categories to be covered by standards promulgated within 2, 4, 7, or 10 years<br>- priorities to consider known or anticipated health and environmental effects, quantity and location of emissions, and potential for grouping categories | - publish schedule, Nov '92 | - screening assessment using available effects and exposure data to assess relative ranking of source categories | RR | a,d |

Table 1: Overview of CAA Title III Risk-Related Requirements

| Statutory Cite/Project Description | Statutory Deadline | Data Considered for Finding | Level of Analysis[1] | Level of Review[2] |
|---|---|---|---|---|
| <u>Section 112(f) Residual Risk</u><br><br>- evaluation of risks associated with emissions following implementation of control technology standards promulgated under Section 112(d)<br>- EPA/Surgeon General report to evaluate methods for evaluating health risks, significance of residual risks, additional control options and associated costs, uncertainties associated with analysis, and recommended legislative changes<br>- default, if Congress does not act on recommended legislative changes, is to use previous methods (e.g., benzene decision) | - EPA/Surgeon General report, Nov '96<br><br>- promulgation of additional standards within 8 years of Section 112(d) standards | - characterization of effect(s) with quantification of exposure and dose-response<br><br>- consideration of individual and population risks | QRA | a,d,e |
| <u>Section 112(g) Modifications</u><br><br>- identification of relative ranking of HAPs based upon potential to elicit health effects considering threshold and nonthreshold effects<br>- ranking to be considered in evaluating emission offsets<br>- establishment of de minimis levels | - guidance, May '92 | - ranking using available effects data to assess relative toxicity of the HAPs and establish de minimis levels | HR | a,c,d,e |

Table 1: Overview of CAA Title III Risk-Related Requirements

| Statutory Cite/Project Description | Statutory Deadline | Data Considered for Finding | Level of Analysis[1] | Level of Review[2] |
|---|---|---|---|---|
| Section 112(i) Early Reduction Program<br><br>- extension of compliance with control technology standards allowed<br>- special consideration of "highly toxic" pollutants | - proposed rule, Jun '91<br>- final rule, Spring '92 | - establishment of highly toxic pollutants similar to Lesser Quantity Emission Rates program identified above | HR | a,c,d,e |
| Section 112(k) Urban Area Source Program<br><br>- development of research program on the sources of HAPs in urban areas<br>- identification of $\geq$ 30 HAPs and associated source categories that pose the greatest threat to public health in urban areas<br>- development of national strategy, accounting for $\geq$ 90% of the identified HAP emissions, resulting in $\geq$ 75% reduction in cancer incidence | - report to Congress on research activities, Nov '93<br>- report to Congress on national strategy, Nov '95<br>- implement strategy such that sources are in compliance, '99 | - analysis of urban exposures using ambient monitoring data and modeling of estimated emissions<br><br>- sufficient data to identify critical effect(s) and dose-response relationships | RR, QRA | a,b,c,e (d, as needed) |

Table 1: Overview of CAA Title III Risk-Related Requirements

| Statutory Cite/Project Description | Statutory Deadline | Data Considered for Finding | Level of Analysis[1] | Level of Review[2] |
|---|---|---|---|---|
| Section 112(m) Great Waters Study<br><br>- investigation of the contribution of atmospheric deposition of HAPs to the Great Lakes, Chesapeake Bay, Lake Champlain, and coastal waters<br>- development of regulations as necessary to prevent adverse health and environmental effects | - establish Great Lakes monitoring network, Dec '91<br><br>- report to Congress, Nov '93<br><br>- promulgation of additional regulatory measures, Nov '95 | - sufficient data required to identify critical HAPs, potential exposures and critical effects<br>- key parameters to consider include: environmental persistence, bioaccumulation potential, delineation of contribution of air emissions vs. other sources of pollutants | RR, QRA | a,b,c,e (d, as necessary) |
| Section 112(n) Electric Utility Study<br><br>- evaluation of public health risks associated with HAPs emitted from electric steam generating units following implementation of Title IV (acid rain) regulations<br>- report on alternative control strategies, if needed | - report to Congress, Nov '93 | - sufficient data to identify critical HAPs, their exposures, potential and initial effects<br>- assess individual and population risk | QRA | a,b,c |

Table 1: Overview of CAA Title III Risk-Related Requirements

| Statutory Cite/Project Description | Statutory Deadline | Data Considered for Finding | Level of Analysis[1] | Level of Review[2] |
|---|---|---|---|---|
| Section 112(n) NIEHS Study of Mercury<br><br>- establishment of threshold concentrations of mercury | - report to Congress, Nov '93 | - sufficient hazard and dose-response data relevant to establishing a threshold concentration for mercury | IIA | b,c (possible) |
| Section 112(n) Mercury Study<br><br>- evaluation of mercury emissions from utilities, municipal waste combustors, and other sources | - report to Congress, Nov '94 | - identify health and environmental effects | RR | a,b |
| Section 112(n) Hydrogen Sulfide Study<br><br>- assessment of public health and environmental hazards associated with emissions from the extraction of oil and natural gas<br>- development and implementation of control strategy, as needed | - report to Congress, Nov '92 | - sufficient evidence on health and environmental effects<br>- adequate emissions data from the oil and natural gas industry | RR | a,b,(d, as needed) |

Table 1: Overview of CAA Title III Risk-Related Requirements

| Statutory Cite/Project Description | Statutory Deadline | Data Considered for Finding | Level of Analysis[1] | Level of Review[2] |
|---|---|---|---|---|
| Section 112(n) Hydrofluoric Acid Study<br><br>- assessment of the potential health and environmental hazards of HF emission releases including worst case accidental releases<br>- recommendation for reducing hazards, if appropriate | - report to Congress, Nov '92 | - sufficient evidence on health and environmental effects<br>- adequate emissions data and probability of accidental release events | RR | a,b,(d, as needed) |
| Section 112(o) National Academy of Sciences Study<br><br>- review of EPA risk assessment methods<br>- exploration of opportunities to improve current methods | - report to Congress, May '93 | - all data considered relevant by the committee | | e |

Table 1: Overview of CAA Title III Risk-Related Requirements

| Statutory Cite/Project Description | Statutory Deadline | Data Considered for Finding | Level of Analysis[1] | Level of Review[2] |
|---|---|---|---|---|
| Section 112(r) Accidental Release Program<br><br>- identification of principal pollutants (≥ 100) and associated threshold quantities<br>- pollutant petition process included<br>- establishment of Chemical Safety Board<br>- development of regulatory program | - report to Congress on use of hazard assessments, May '92<br>- promulgation of pollutant list and threshold quantities, Nov '92<br>- report to Congress on regulatory recommendations, Nov '92 | - identification of potential for death, injury, serious adverse effect(s) | QRA | a,d,e |

[1] Levels of Anaylsis: HA, Hazard Assessment; HR, Hazard Ranking; RR, Risk Ranking; QRA, Quantitative Risk Assessment, as described in Section I.C.
[2] Levels of Review as described in Section I.D

## II. Question 2: What has EPA done in the past toward those or similar risk assessment requirements, and why did EPA take the specific actions it did?

### II.A Introduction

The following sections describe the framework for risk assessment presented by specific activity. The first section describes these activities generically, and subsequent sections provide examples of past and current or planned assessments.

### II.B Generic Discussion

The approach EPA follows in conducting risk assessments follows the framework proposed by the National Research Council (NRC) of the National Academy of Sciences in 1983. This process was described in a book entitled " Risk Assessment in the Federal Government: Managing the Process" and identified risk assessments as containing one or more of the following four components: hazard identification, dose-response assessment, exposure assessment, and risk characterization.

In response to the NRC proposal, EPA issued several risk assessment guidelines addressing such areas as carcinogenicity, developmental toxicity, chemical mixture assessment, reproductive toxicity, exposure assessment, and mutagenicity. The EPA is continuing to develop guidelines to address various issues including risk assessment methods for evaluating noncancer effects, e.g, guidelines discussing immunotoxicity and respiratory toxicity.

The sections that follow generally discuss the process of developing risk assessment guidelines and provide examples of the efforts undertaken by the Agency to address the four components of the risk assessment process. For instance, the hazard identification and dose-response assessment steps are incorporated into the development of the hazard assessment documents.

### II.B.1 Risk Assessment Guideline Development

The EPA has published guidelines addressing various aspects of risk assessment to direct the Agency in the consistent evaluation of environmental pollutants. The process of developing Agency-wide risk assessment guidelines is a multi-year procedure incorporating the state-of-the science with both internal and external expertise. This process is illustrated in Figure 3. The guidelines serve two purposes: (1) to guide EPA scientists in conducting Agency risk assessments and (2) to inform EPA decision makers and the public about these procedures. The principles set forth in the EPA risk assessment guidelines apply across all risk-based decisions considered by the Agency.

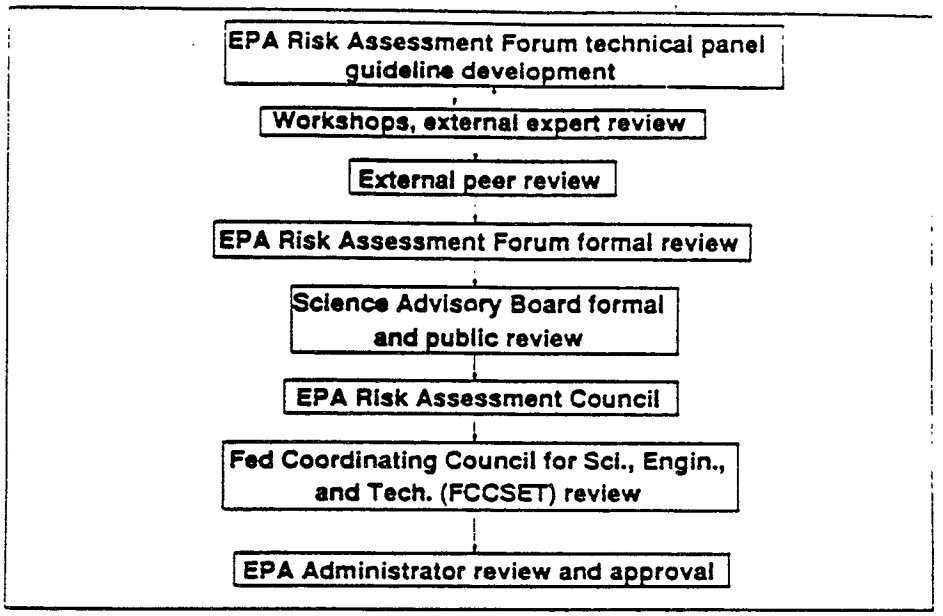


Figure 3: Risk Assessment Guidelines Development Process

The emphasis of these guidelines is that the risk assessments should be conducted on a case-by-case basis considering all relevant information. The information considered includes: the level of analysis required to meet the needs of the risk manager, the availability of data, and the existing methods for appropriately interpreting the scientific data. The guidelines also stress the need to clearly articulate the scientific basis and rationale for each assessment along with its associated strengths and weaknesses. Included must be a description of the uncertainties, assumptions, and limitations of the risk assessment conducted.

II.B.2 Hazard Assessment Document Development

Hazard Assessment Documents (HADs) were commissioned at the request of EPA's Office of Air Quality Planning and Standards (OAQPS) to provide health information on the 30+ substances that were being considered for listing under section 112 of the Clean Air Act in the 1980's. In 1982, an EPA Office of Research and Development (ORD) committee was convened for the purposes of developing a plan for producing hazard assessment documents. They were specifically charged with determining the scope and content of the documents, procedures for production and peer review, and the schedules and resources necessary for production within the anticipated deadlines.

The most immediate purpose of the documents was to meet the needs of OAQPS by providing critical evaluations of all the pertinent health literature and data to determine whether or not significant human health effects were associated with exposure to chemicals at ambient air concentrations. The committee agreed these should focus on air-related health concerns, but attempts would be made to identify other EPA program offices as potential users, requiring the structure of the documents to consider multi-media assessments. The contents of each document would consider:

- physical and chemical characteristics
- man-made and natural sources and emissions
- environmental distribution and measurement, including measurement techniques, transport and fate, environmental concentrations and exposures (multi-media)
- ecological effects
- biological disposition, metabolism, and pharmacokinetics
- toxicological overview of health effects
- specific health effects, i.e., mutagenicity, carcinogenicity, and other noncancer health effects
- synergism and antagonism
- health risk information

A multi-tiered assessment approach was employed, with sucessively more detailed and extensive assessments conducted as warranted by preceding outcomes. The results of each level would be reviewed by the program office (OAQPS) and considered along with exposure assessment information developed by OAQPS in order to determine the neccesity for further, more detailed assessment. The process is diagrammed below in Figure 4.

## II.B.3 Exposure Methodology

The first systematic exposure assessments of hazardous air pollutants (HAPs) began as a result of provisions in the Clean Air Act of 1970 requiring the identification and listing of HAPs, as well as the promulgation of emissions standards for those listed HAPs. To assist in these assessments, the Human Exposure Model (HEM) was developed by OAQPS for use as a screening model in the identification and national assessment of candidate HAPs. This role expanded in the early 1980's to include more detailed quantitative evaluation of health risks (principally cancer) associated with stationary emission sources of HAPs.

In 1986, EPA published guidelines on conducting exposure assessments. The guidelines were developed to assist future assessment activities and encourage improvement in those EPA programs that require, or could benefit from, the use of exposure assessments. The authors of the guidelines also attempted to promote

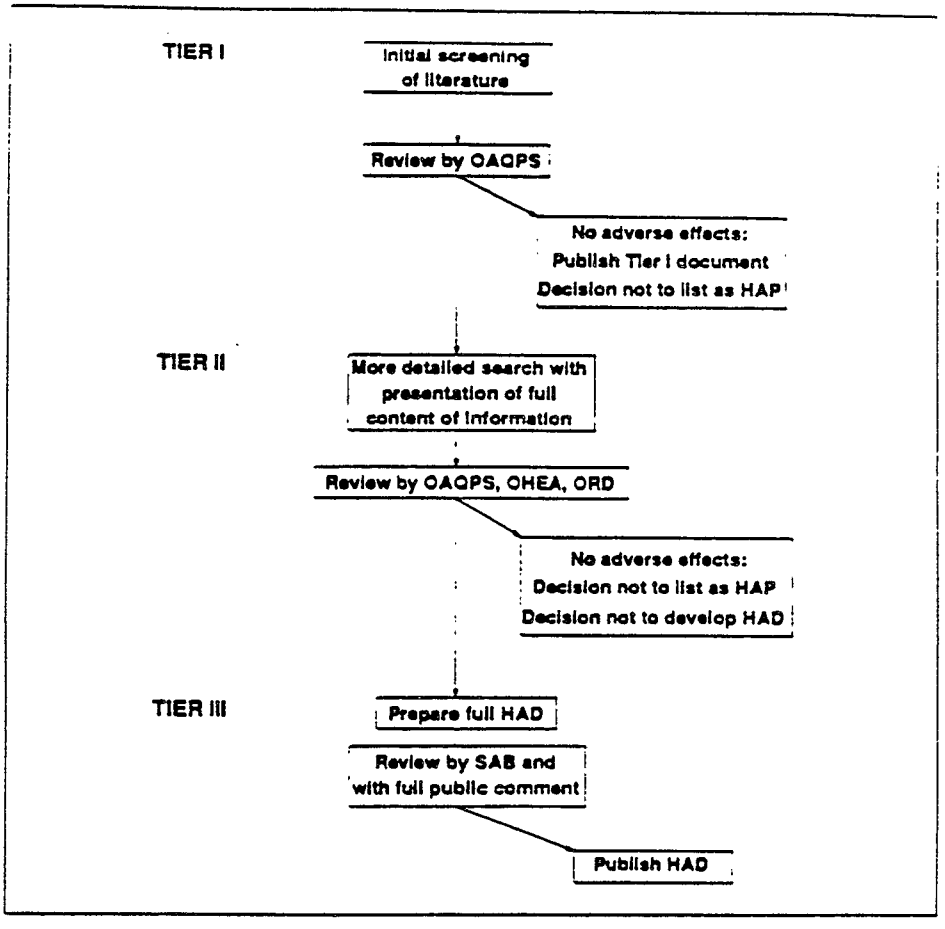


Figure 4: Hazard Assessment Document Development

consistency among various exposure assessment activities that are carried out by the Agency. The guidelines recognized that the main objective of an exposure assessment is to provide reliable exposure data or estimates for a risk characterization. Since a risk characterization requires coupling exposure information and toxicity or effects information, the exposure assessment process should be coordinated with the effects assessment. The OAQPS has interpreted this important consideration to mean a balancing of uncertainties in the exposure assessment with the uncertainties in the effects assessment, i.e., quality toxicity assessments are supported with quality exposure assessments. In 1991, EPA revised the exposure assessment guidelines to substantially update the earlier guidelines. The new guidelines incorporate

developments in the exposure assessment field since 1986, both including the previous work and adding several topics not covered previously. The EPA will be examining the exposure assessment process for HAPs to ensure continuity with the new guidelines. Figure 5 presents a diagram of the process.

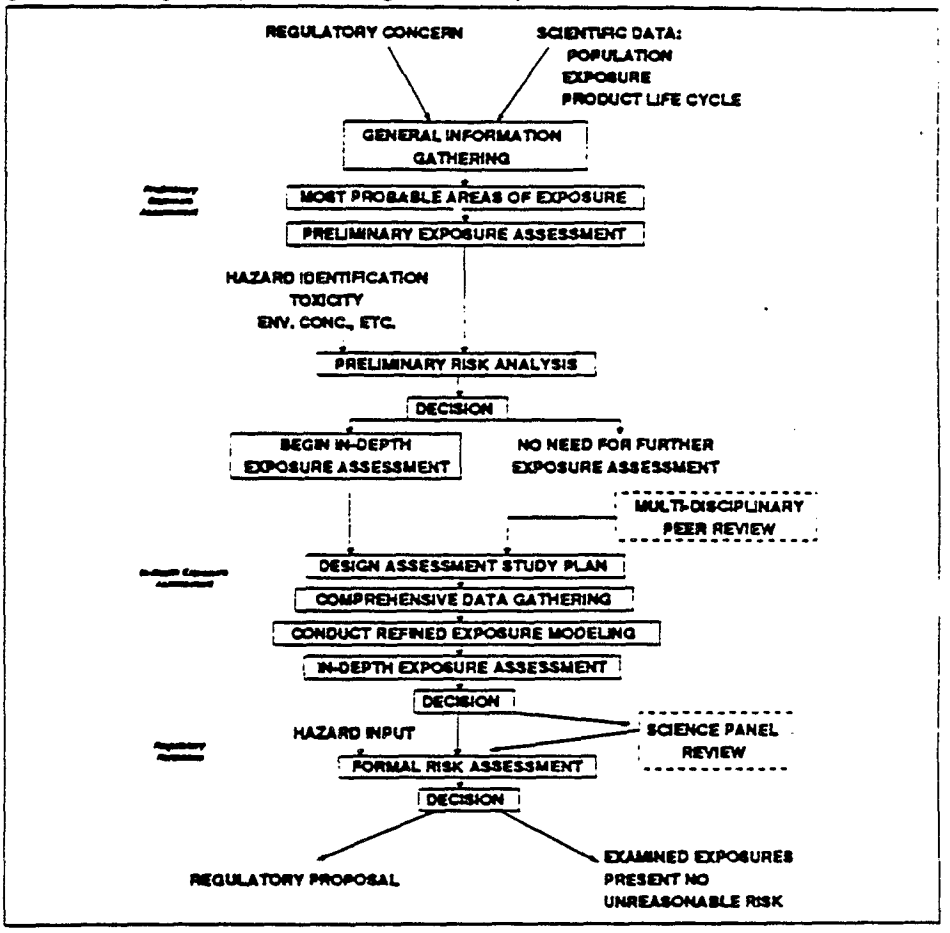


Figure 5: Integration of Exposure Assessment into Risk Assessment Process

## II.B.4 Risk Characterization and Treatment of Uncertainty

One of the issues which EPA continues to address has been the characterization and communication of estimated risks and their uncertainties to a variety of audiences, including Agency risk managers, State and local air pollution control agencies, the

public, the affected industries, environmental groups, and other interested parties. The OAQPS traditionally conducted risk characterizations nationally by source category, rather than presenting risks posed by each emission point or facility. In the early 1980's risk estimates were used largely to rank source categories by their estimated potential risks. As experience was gained with risk assessments and the perceived need of risk managers to have more information to make more informed decisions increased, the national source category approach evolved into plant-by-plant and, in some cases, emission point-by-emission point analyses.

The process of risk characterization combines the results of the hazard identification, dose-response assessment, and exposure assessment. In evaluating HAPs, EPA reviews the available information and determines the most appropriate level of risk estimation that may be conducted using these data. The data generally can be categorized into four areas: (1) source and emissions; (2) transport of the pollutant from the source to the target population; (3) exposure of the target population; and (4) adverse effects resulting from the exposure. Depending upon the quantity and quality of the data, the risk assessment may be qualitative and/or quantitative in nature.

Qualitative risk assessments include an analysis of the existing data base and the potential for the pollutant to elicit an effect in a population. This assessment may involve the classification of the data into weight-of-evidence categories and would include a consideration of the severity of the effect anticipated in the exposed population.

Quantitative cancer risk assessments have frequently included the presentation of information in three ways: (1) estimated population risk, expressed as average annual incidence; (2) maximum individual lifetime risk; and (3) distribution of individual risk across the exposed population, i.e., the number of individuals at risk in various risk intervals (e.g., $10^{-4}$, $10^{-5}$, $10^{-6}$).

The evaluation of potential noncancer risks has frequently involved the comparison of estimated ambient levels with a reference level. For example, the risk for developmental toxicity may be inferred by comparing the reference dose for this effect ($RfD_{DT}$) and the human exposure estimate or by calculating the "margin of exposure" (MOE). The $RfD_{DT}$, derived by applying uncertainty factors to the no observed adverse effect level (NOAEL) (or the lowest observed adverse effect level, LOAEL), differs from the RfD because the former is based on a short duration of exposure rather than chronic exposure situations. The MOE is the ratio of the NOAEL from the most appropriate or sensitive species to the estimated human exposure level, and is presented along with a discussion of the weight-of-evidence (WOE) classification. The WOE incorporates information from all relevant studies and represents a judgment based on the collective database as to the likelihood that exposure to a specific substance may pose a risk to humans. Placing an agent in a

particular WOE category such as "adequate evidence for human developmental toxicity" does not mean that it will be a developmental toxicant at every dose, since the Agency assumes the existence of a threshold for the effect. Appendix A presents additional information on EPA's risk assessment guidelines for developmental toxicity.

As tools develop in the area of noncancer risk assessment along with expansion of existing data bases, quantitative presentation of risk assessments similar to analyses conducted for potential carcinogenic risks may be possible. It should be noted that the presentation of either a qualitative or quantitative risk assessment must always be accompanied by a description of the limitations associated with the analysis including attendant assumptions and uncertainties.

As risk managers seek to derive the maximum information possible for decision-making, greater emphasis has been placed on the characterization of uncertainty. The key uncertainties associated with the overall risk assessment process can be divided into three areas: uncertainties in the quantification of health effects; uncertainties in modeling the atmospheric dispersion of emitted HAPs; and uncertainties in the assessment of population exposure. Uncertainties associated with health effect quantification arise from use of the linear multi-stage model for estimating cancer potency, extrapolation from high-dose to low-dose, extrapolation from animal to sensitive human populations and extrapolation across various routes of exposure. A critical need is to expand our understanding about the relevant underlying physical, chemical, and biological mechanisms that affect the validity of extrapolation assumptions. Uncertainties associated with atmospheric dispersion modeling stem from uncertainties in emission rates, meteorological and terrain information, and relation of assumed stack parameters and locations to actual values. Uncertainties in the assessment of population exposure arise from uncertainties in the location and activity patterns of exposed populations, duration of actual exposures in each microenvironment, and extrapolations across exposure conditions. Appendix P includes a chart which illustrates the expected magnitude of uncertainties surrounding several exposure parameters evaluated in the assessment of benzene emissions. Activities within EPA to reduce the uncertainties in each of these areas are described later in the section on evolution of exposure and risk assessment methodologies (II.D.6).

### II.B.5 Some Differences Between Past and Present Risk Assessment

The new CAA expands the scope of air toxics regulations. Consequently, expectations at each level of assessment have increased. For example, hazard assessment and hazard ranking currently place greater focus on the relative hazard and potency of the effects. Exposure information and emission data are also subject to this increased level of need. For example, the Toxic Release Inventory (TRI) data base, established under Section 313 of the Superfund Reauthorization and Recovery Act (SARA), contains emission data on many HAPs, but the data are generally not

sufficient to use in quantitative risk assessments. The data base is limited in that it only covers certain industrial types, and is required only for relatively large plants. While this type of information may have been useful for defining a problem or to derive crude estimates of exposure in the past, it is not anticipated to be sufficient for quantitative risk assessments.

The questions and needs to be addressed under the CAA go beyond the data issue. The assessment procedures of the past will have to be reexamined in light of the new legislation. Requirements associated with the residual risk determinations bring about additional concerns for the quantitative risk assessment process. Some of these concerns are:

- assessing residual risk from multiple pollutants rather than individual pollutants within a source category
- determining the approach appropriate to evaluating risk to the most exposed individual
- assessing noncancer health risks
- determining the risks from less than chronic exposure, especially acute exposures
- factoring population mobility and activity patterns into the risk assessment process
- identifying sensitive populations
- assessing ecological risks

While these may not be new concerns, the CAA of 1990 has focused greater attention on these issues.

## II.C Examples of Past Assessments

### II.C.1 Problem Definition

Exposure to HAP emissions may result in a variety of adverse health effects considering both cancer and noncancer endpoints. In an effort to better understand the "big picture" of hazardous air pollutant exposures, EPA undertook broad, screening studies in the 1980's to evaluate the releases of these pollutants and the relative implication of the resulting exposures to human health.

One study, entitled "Cancer Risks from Outdoor Exposure to Air Toxics" (Appendix B), assessed the magnitude and nature of potential cancer risks associated with exposure to hazardous air pollutants. Originally conducted in 1985 and updated in 1990, the work broadly assessed long-term exposures to HAPs and estimated potential cancer risks associated with these pollutants. The results of the updated analysis estimated an increase of cancer cases to be between 1700 and 2700 per year as a result of HAP exposure. Approximately 40 percent of these cases were associated

with emissions from stationary sources versus mobile sources. In addition, maximum individual cancer risks were estimated to be in excess of 1 in 1,000 at several locations.

## II.C.2 Hazard Assessment

A hazard assessment as defined by EPA guidelines is an evaluation of a chemical's toxicity and potential to cause adverse health and environmental effects. At minimum, it entails a search of the scientific literature and an assessment of the amount and quality of the data including the availability of dose-response data.

A qualitative assessment of data includes evaluation of available human, animal, and in vitro evidence in determining how likely a chemical is to elicit an adverse effect in humans or other exposed populations of interest. This type of information is generally examined within the framework of a weight-of-evidence classification scheme.

If sufficient quantitative data are available, a dose-response assessment may be conducted. For carcinogens, the Agency has traditionally developed unit risk estimates (UREs) to express the relationship between dose and carcinogenic response. An URE, under assumption of low-dose linearity, is an estimate of the excess, lifetime risk due to continuous exposure to one unit of concentration (e.g., $ug/m^3$ for inhalation). For noncarcinogens, limited data and risk assessment methods allowed only the identification of effect levels rather than a quantitative expression of the data.

In addition to toxicity data, other information that is typically included in a hazard assessment include data on a chemical's environmental fate, transport, or persistence in the environment. If the data are sufficient, a hazard assessment presents a profile of a chemical's toxicity, potential health and environmental risk, and related chemical characteristics. In practice, this is best exemplified by HADs (see discussion in Section II.B.2). The HADs incorporate all of the information listed above. These documents also undergo a peer-review by EPA's Science Advisory Board (refer back to Figure 4). This type of assessment formed the principal basis for decisions to list chemicals as HAPs under the previous Section 112.

## II.C.3 Hazard Ranking

There are no past examples of hazard ranking. Rankings that were done used emission data to rank rather than toxicity data which, for the most part, lacked sufficient potency data to do adequate ranking.

II.C.4 Risk Ranking

Figure 6 illustrates the process used to identify HAPs prior to passage of the Clean Air Act Amendments of 1990. During the mid-1980's, the Agency modified this process to add in the "Intent-to-List" procedure prior to actually listing a chemical under Section 112 of the CAA. Table 2 identifies the pollutants that EPA formally evaluated during this time frame and the resulting decision to continue analysis (intent-to-list) or discontinue analysis (not-to-regulate). Examples of the notices published in the Federal Register are included in Appendix C.

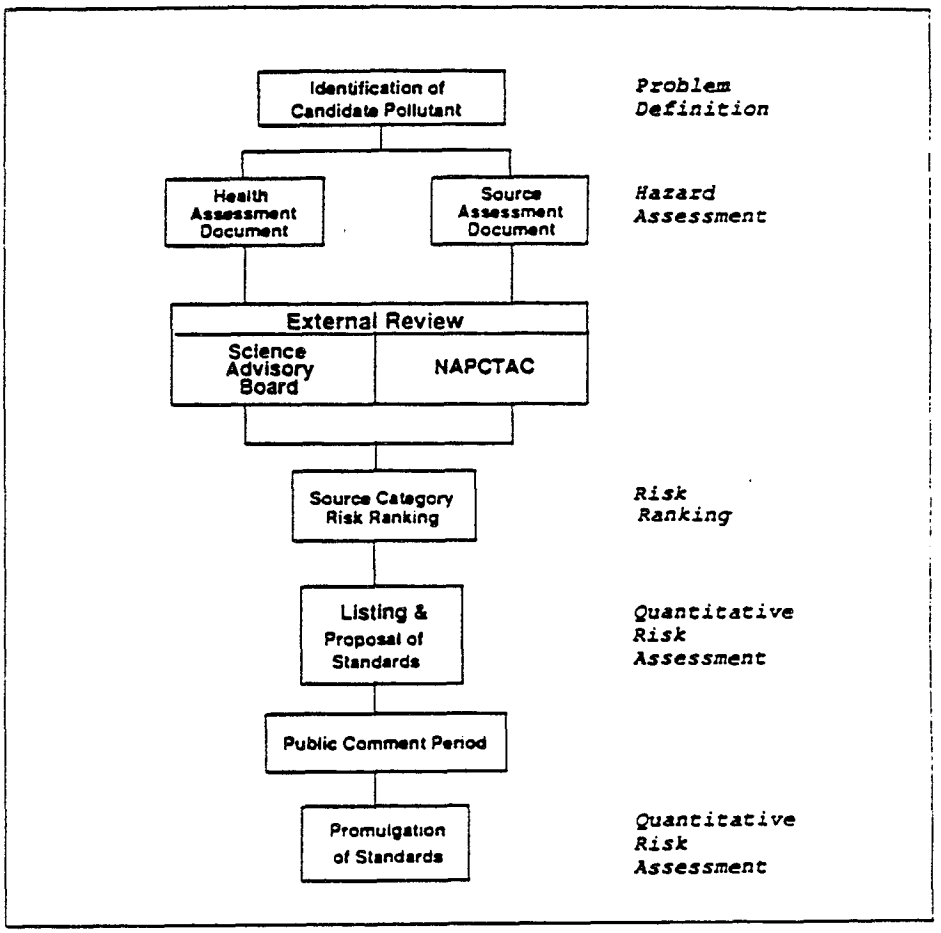


Figure 6: Identification, Assessment, and Regulation of HAPs

Table 2: 1984-87 Hazardous Air Pollutant Decisions

| POLLUTANT | ACTION | CITATION |
|---|---|---|
| Acrylonitrile | State Referral | 50FR24319; June 10, 1985 |
| 1,3-Butadiene | Intent-to-List | 50FR41466; October 10, 1985 |
| Cadmium | Intent-to-List | 50FR42000; October 16, 1985 |
| Carbon Tetrachloride | Intent-to-List | 50FR32621; August 13, 1985 |
| Chlorofluorocarbon 113 | Not-to-Regulate | 50FR24313; June 10, 1985 |
| Chlorinated Benzenes | Not-to-Regulate | 50FR32628; August 13, 1985 |
| Chloroform | Intent-to-List | 50FR39626; September 27, 1985 |
| Chloroprene | Not-to-Regulate | 50FR39632; September 27, 1985 |
| Chromium | Intent-to-List | 50FR24317; June 10, 1985 |
| Coke Oven Emissions | Listing Notice | 49FR36560; September 18, 1984 |
| Copper | Not-to-Regulate | 52FR5496; February 23, 1987 |
| Epichlorohydrin | Not-to-Regulate | 50FR24575; June 11, 1985 |
| Ethylene Dichloride | Intent-to-List | 50FR41994; October 16, 1985 |
| Ethylene Oxide | Intent-to-List | 50FR40286; October 2, 1985 |
| Hexachlorocyclopentadiene | Not-to-Regulate | 50FR40154; October 1, 1985 |
| Manganese | Not-to-Regulate | 50FR32627; August 13, 1985 |
| Methyl Chloroform | Not-to-Regulate | 50FR24314; June 10, 1985 |
| Methylene Chloride | Intent-to-List | 50FR42037; October 17, 1985 |
| Municipal Waste Combustion Emissions | Advance Notice of Proposed Rule-making | 52FR25399; July 7, 1987 |
| Naphthalene | Not-to-Regulate | 53FR9138; March 1, 1988 |
| Nickel | Not-to-Regulate | 51FR34135; September 25, 1986 |
| Perchloroethylene | Intent-to-List | 50FR52880; December 26, 1985<br>*51FR7719; March 5, 1986 |
| Phenol | Not-to-Regulate | 51FR22854; June 23, 1986 |
| Polycyclic Organic Matter | Not-to-Regulate | 49FR31680; August 8, 1984 |
| Toluene | Not-to-Regulate | 49FR22195; May 25, 1984 |
| Trichloroethylene | Intent-to-List | 50FR52422; December 23, 1985<br>*51FR7714; March 5, 1986 |
| Vinylidene Chloride | Not-to-Regulate | 50FR32632; August 13, 1985 |
| Zinc/Zinc Oxide | Not-to-Regulate | 52FR32597; August 28, 1987 |

* Clarification Notice

II.C.5 Quantitative Risk Assessment: The Regulation of Benzene: 1977-1989

**Note: The following sections presen an overview of the evolution of risk-based decision-making under the old Section 112, using the regulation of benzene as the principal example. The text is supplemented by several appendices that provide examples of decision documentation and briefing materials from these periods.**

Introduction

In June of 1977, EPA added benzene to the list of HAPs under Section 112. For the next twelve years, under a succession of 6 Administrators, the air program wrestled with the regulation of a known human carcinogen for which a health effect threshold could not be established, under an authority requiring the protection of public health with an ample margin of safety. During this period, benzene became the test case for a series of procedural interpretations and re-interpretations of the statutory language, culminating in the 1987 vinyl chloride opinion by the D.C. Court of Appeals (NRDC v. U.S. EPA, July 28, 1987) and the revision of the statute in the 1990 amendments to the Clean Air Act.

The regulation of benzene also spans a period during which the methods for quantitatively estimating risks from exposure to airborne carcinogens evolved, and the appropriate role of such estimates in the decision process was hotly debated within, as well as outside, the EPA. For these reasons, benzene represents an interesting and illuminating case study of quantitative risk assessment and its use in determining the appropriate level of control under Section 112. A chronology of EPA's regulatory policy under Section 112 is summarized in Figure 7.

Benzene and the Airborne Carcinogen Policy (1977-1983)

The EPA listed benzene as a HAP in 1977 based on growing evidence of a link between occupational exposure and an increase in the incidence of acute myelogenous leukemia (Appendix D - Benzene Listing). Prior to the listing of benzene, EPA had regulated four pollutants under Section 112: asbestos, beryllium, and mercury in 1971; and vinyl chloride in 1974-75. In the absence of procedures for estimating cancer risk, the original asbestos standard was based on "no visible emissions". Beryllium had not been identified as a carcinogen (berylliosis was the effect of concern) and the toxic effects of mercury were addressed with an ambient air guideline, taking into consideration exposure by other routes (e.g., ingestion).

By the listing of vinyl chloride in 1974, quantitative techniques were under development within the EPA. In conjunction with the promulgation of the vinyl

| Era | | Approach |
|---|---|---|
| 1971 | Asbestos | "No Visible Emissions" |
| | Beryllium | Best Technology |
| | Mercury | Ambient Guideline |
| 1974-75 | Vinyl Chloride | Best Available Technology (BAT) |
| 1977-81 | Benzene/Carcinogen Policy | BAT/Beyond BAT |
| 1983-84 | Risk Management | Weigh All Factors |
| 1987 | Vinyl Chloride Opinion | "Safe"/Ample Margin Of Safety |
| 1988 | Benzene Proposal | "Framing the Debate" |
| 1989 | Benzene Promulgation | "Fuzzy Bright Line" |
| 1990 | CAA Amendments | MACT Now/Residual Risk Later |

Figure 7: Chronology of Section 112 Regulatory Policy Development

chloride emission standards, rough estimates of projected incidence of angiosarcoma were made, but were considered too uncertain to be used in the determination. The vinyl chloride standards were principally based on the application of the best available control technology (BAT).

In May of 1976, EPA issued the first carcinogenicity guidelines (Appendix E - "Health Risk and Economic Impact Assessments of Suspected Carcinogens"). In the benzene listing notice the following year, EPA announced the conduct of a benzene health risk assessment and indicated that the "relative risk to the public" would be considered in judging "the degree of control which can and should be required". The risk assessment, containing the original unit risk estimate for benzene, was subsequently published in January 1979 (Appendix F - Benzene Population Risk).

The advent of a quantitative methodology and external pressure for a more aggressive program under Section 112 led to the development of EPA's airborne carcinogen policy. The policy was published in October 1979, as a proposed interpretive rule outlining procedures for the identification, assessment, and regulation of airborne carcinogens emitted from stationary sources (Appendix G - Airborne

Carcinogen Policy). The policy reflected a technology-based approach to emission standard development with a limited role for quantitative risk assessment in establishing priorities and ensuring that the residual risks following the application of BAT were not unreasonable. The first round of benzene standards, beginning with the regulation of maleic anhydride plants in 1980, followed the proposed procedures, the shorthand for which became "BAT/Beyond BAT". Although a final version of the proposed policy was prepared in 1981, incorporating public comments, the policy was never promulgated. The procedures were informally followed, however, up to the introduction of the "risk management" approach in 1983.

Also in 1979, the development of the Human Exposure Model (HEM) (Appendix H - HEM Description) provided a means of estimating and summing ambient exposures across the populations living in the vicinity of emitting sources. These estimates were then combined with the unit risk estimate to yield cancer risk estimates. In the first benzene standards, estimates of maximum individual lifetime risk and annual incidence were calculated. The risk estimates were sometimes displayed as small ranges, incorporating some of the quantifiable sources of uncertainty. Other uncertainties were usually presented as tabular footnotes (Appendix I - Proposed Maleic Anhydride Standards) .

The Risk Management Era (1983-1985)

The change of Administrations in 1981 brought an increasing emphasis on the cost-effectiveness of regulation and regulatory reform. In this light, the presumption expressed in the proposed carcinogen policy - that, given the uncertainty in risk estimation, significant source categories of airborne carcinogens should be regulated, at a minimum, to a level of control constituting BAT - was called into question. The re-examination of this presumption resulted in a revised policy which held that risk information, as well as other relevant factors, should be considered in determining the appropriate level of control, including finding that control was unwarranted. One result of this change was to place greater weight on the risk assessment in the decision process.

In 1984, after "weighing all factors", EPA made several changes to the proposed benzene rules, including withdrawal of the maleic anhydride proposal, arguing that the risks were "too small to warrant Federal regulatory action" (Appendix J - Withdrawal of Proposed Standards). These decisions were promptly challenged by the NRDC, arguing the uncertainties in the risk estimates and the inappropriate consideration of cost in regulatory decisions made under Section 112. The issues raised were similar to litigation already pending on amendments to the original vinyl chloride standards.

Also during 1984, work was begun to revise the benzene unit risk estimate, based on new human and animal data and an improved methodology. A revised

estimate was transmitted to the air program by the Office of Research and Development in early 1985 (Appendix K - "Interim Quantitative Unit Risk Estimates").

The Vinyl Chloride Opinion (1987)

On July 28, 1987, Judge Robert Bork, writing for the D.C. Circuit Court of Appeals, remanded the vinyl chloride amendments to EPA, finding that the Agency had placed too great an emphasis on technical feasibility and cost rather than the provision of an "ample margin of safety" as required by the statute (Appendix L - Vinyl Chloride Opinion). The opinion also laid out a process for making decisions, consistent with the requirements of the law.

The Bork opinion held that, in setting standards under Section 112, EPA must first determine a "safe" or "acceptable" level, and that this level must be established considering only the potential health impacts of the pollutant. Once an acceptable level was identified, the level could be reduced further, as appropriate and in consideration of other factors, including cost, technical feasibility, affordability, etc., to provide the required ample margin of safety. The Court also held, however, that "safe" did not require a finding of "risk-free" and that EPA should recognize that activities such as "driving a car or breathing city air" may not be considered "unsafe".

Benzene Proposal (1988)

The EPA accepted voluntary remand of the 1984-85 standards and issued a new proposal in July 1988, consistent with the vinyl chloride opinion. Given the requirement for a determination of "safe", the importance of the quantitative risk assessment took on even greater emphasis. This is evident in the senior management briefings on the proposal (Appendix M - Briefing for the Administrator). The determination of a "safe" or "acceptable risk" level continued to be problematic, however, in part due to the diversity of opinion within, and external to, the Agency on what constituted an "acceptable risk" but, also to the dicta of the legal opinion itself. The decision appeared to accept "driving a car or breathing city air" as examples of activities judged to be safe by society. This raises the issue of whether society's judgment to drive or live in cities is founded solely on the possible health impacts of these activities, rather than a consideration of all factors, which would be prohibited in the EPA framework.

Several options for the determination of "acceptable" risk were considered in the months preceding proposal. The preferred option, a case-by-case consideration of all of the relevant health information was described in a memorandum by the Administrator (Appendix N - "Proposed Benzene NESHAP Decisions"). Ultimately, however, EPA proposed four options for the determination of "safe", "framing the debate" for public comment (Appendix O - Proposed Benzene NESHAP). With the

exception of the case-by-case alternative, the options represented bright line risk targets, either individual or population risk. All factors were to be considered in the determination of the ample margin of safety.

Benzene Promulgation (1989)

The EPA received a large volume of comments on the proposed rules. Again, the risk methodology and estimates, and the proposed acceptability criteria were extensively discussed. The appended briefing for the Assistant Administrator (Appendix P - "Consideration of Comments") illustrates the emphasis on the risk methods and underlying uncertainties. During this time, there was also increased interest in not only the estimates of maximum individual and population risk, but also the distribution of individual risk across the exposed population.

In September of 1989, EPA promulgated emission standards for several categories of benzene sources (Appendix Q - Final Benzene Rules). The decision criteria adopted represented a blend of several of the proposed options. The EPA argued for the consideration of all relevant health information and established "presumptive benchmarks" for risks that would be deemed "acceptable". The goal, which came to be known as the "fuzzy bright line", held that risks would be deemed acceptable if few, if any, individuals were exposed above a 1 in 10,000 lifetime cancer risk, and, as much of the exposed population as possible was below a lifetime risk of 1 in 1,000,000.

The selection of even "fuzzy" risk targets placed greater emphasis on the development and communication of risk characterization results. For the final benzene rules, this was evident in the decision briefings as well as the development of question and answer materials (Appendix R - Benzene Questions and Answers) and the decision to provide advance briefings for the news media (Appendix S - Background Information for the Media).

The Clean Air Act Amendments (1990)

The amendments to Section 112 require the application of technology-based standards to major and designated area source categories as a first step. Following compliance with the maximum achievable control technology (MACT) standards, EPA is required to evaluate residual risks, applying the decision criteria used in the final benzene rules, to determine whether the technology-based rules provide an ample margin of safety to protect public health. Risk assessment will continue to play an important role in the implementation of this and other provisions of Section 112 and the importance of appropriate methodologies and characterization of uncertainties cannot be understated.

## II.D Examples of Present Assessments

### II.D.1 Problem Definition

Sections 112(c) and (k) of Title III prescribe an Urban Area Source program that includes the development of a national strategy requiring 75% or more reduction in cancer incidence associated with emissions of 30 or more HAPs that "present the greatest threat to public health in the largest number of urban areas". For this national strategy to be implemented, many issues need to be defined and addressed including:

- types of sources covered
- selection of the urban areas covered
- selection of the 30 or more HAPs to be regulated on a variety of endpoints and characterization of their ambient levels
- characterization of the emission release parameters
- establishment of an emissions inventory system to help demonstrate that the goals of the strategy are being met
- role of atmospheric transformation

This program requires policy decisions as well as research decisions to be made so that the goal of listing the sources in 1995 and promulgating the subsequent standards for affected sources can be met.

The Great Waters Study (Section 112(m)), requires that EPA, in cooperation with the National Oceanic and Atmospheric Administration, identify and assess the extent of atmospheric deposition of HAPs to the Great Lakes, Chesapeake Bay, Lake Champlain, and coastal waters. A report to Congress is due within 3 years of enactment and biennially thereafter. A plan is being developed to evaluate the information available, the information needed, and how to acquire that additional information. The Report to Congress requires the following information:

- contribution of atmospheric deposition to total pollution loading
- environmental and public health effects
- sources of the pollutants
- contribution of HAPs to water quality violations

To accomplish this, it will be necessary to:

- conduct atmospheric deposition monitoring for source identification and model validation
- conduct atmospheric transport and deposition modeling to include direct and indirect pathways
- develop emission inventories as input to models
- evaluate adverse effects of air toxics on public health and the environment

The Great Waters work shall also support data sharing and the development of remedial action plans (RAPs) and lakewide management plans (LaMPs). The final results of this study may be the promulgation of further emission standards or control measures as may be necessary and appropriate to prevent the adverse effects from occurring.

## II.D.2 Hazard Assessment

With passage of the new CAA, the emphasis on hazard assessment changed from the generation of HADs to the generation of dose-response or potency based estimates where the data supported such an analysis. In selecting appropriated toxicity information, the data required for the statutory findings and mandated deadlines were considered including information on cancer and noncancer effects. For carcinogenic risks, emphasis to date has focused on existing quantitative assessments including unit risk estimates (UREs) (see discussion in Section II.C.2) and $ED_{10}$s.

The assessment of ambient concentrations of HAPs in relation to their potential to elicit adverse noncancer effects presents several challenges. Considerations must include: evaluation of short-term as well as long-term exposures, incorporation of severity-of-effect data, and consideration of reversible versus irreversible effects. The endpoints that may be of most concern could include respiratory effects, developmental/reproductive toxicity, and neurotoxicity.

The quantification of noncancer risks from exposure to inhaled hazardous air pollutants currently focuses on the derivation of inhalation reference concentrations (RfCs). The RfC is defined as an estimate (with uncertainty) of the concentration that is likely to be without appreciable risk of deleterious effects to the exposed population after continuous, lifetime exposure. The RfC focus is on the most sensitive members of the population who may be exposed and the respiratory system as the portal of entry. An experimental exposure level representing the highest level tested at which no adverse effect was observed (NOAEL) is selected from a given study and converted to a human equivalent concentration ($NOAEL_{HEC}$). The critical toxic effect used is the one generally characterized by the lowest NOAEL. This approach is based on the assumption that if the critical toxic effect is prevented, then all toxic effects are prevented. The RfC is derived from the ($NOAEL_{HEC}$) by the application of uncertainty factors to account for extrapolations that may be made. These estimates along with UREs are reviewed within the Agency before incorporation onto EPA's Integrated Risk Information System (IRIS).

Under Title IX of the CAA, EPA is required to develop environmental health assessments for the HAPs. In addition to hazard assessment information, these profiles are to identify data gaps and, where appropriate, identify the additional activities needed to better characterize the "types or levels of exposure which may

present significant risk of adverse effects in humans."

**NOTE: Information concerning revisions to the current EPA's cancer risk assessment guidelines will be provided under separate cover.**

## II.D.3 Hazard Ranking

A further step in the assessment process is the ranking of HAPs based on their relative hazard to human health. The data needs to be collected in a form which allows the comparison of chemical hazards, e.g. comparing similar endpoints of concern. Ideally, the ranking would rely on Agency-reviewed, benchmark risk values such as UREs or RfCs. In reality, due to the lack of health data, the ranking of chemicals may have to rely on less rigorously reviewed values and many assumptions or defaults. The ranking of HAPs for the purpose of offsets under the modifications section of the CAA (Section 112(g)) provides an example of one approach EPA has taken. This section of the CAA requires that EPA issue guidance which includes the ranking of threshold and non-threshold pollutants. Without sufficient data to the contrary, the EPA currently considers all noncarcinogens as threshold pollutants and carcinogens as non-threshold pollutants. As data become available, this general categorization may change for specific pollutants.

The ranking methodology currently being considered under Section 112(g) uses methods already in place, i.e. for establishing Reportable Quantities under the Comprehensive Emergency Response and Compensation Liability Act (CERCLA). Non-threshold pollutants (carcinogens) are ranked by comparing potency estimates ($1/ED_{10}$) and weight of evidence classification. The $ED_{10}$s are defined as the estimated dose associated with a lifetime increased cancer risk of 10%. Threshold pollutants are ranked by either their composite scores (CS) which reflect chronic toxicity, or their level of concern which reflects acute toxicity. Composite scores consider dose-response and severity of effect. The magnitude of the CS determines the ranking position of the chemical (pollutants with large composite scores elicit severe effects at low doses). Under section 112(g), increases in emissions of non-threshold pollutants cannot be offset by decreases in emissions of threshold pollutants, but the reverse is true. The ranking must provide a comparison of the relative hazard within categories of non-threshold and threshold compounds. It is also known that certain pollutants may cause severe effects resulting from acute exposures, therefore the guidance also provides a category for "high concern" threshold pollutants. These pollutants are considered (for the purposes of this section) more hazardous than threshold pollutants but no comparison can be made between these and non-threshold pollutants. If pollutants do not have adequate data to be ranked as a threshold, nonthreshold, or "high concern" pollutant, then that pollutant is considered not tradeable under this section. The general methodology that has been developed to date was reviewed by the Science Advisory Board and National Air Pollution Control

Techniques Advisory Committee (NAPCTAC) while the application undergoes rounds of internal review and public comment.

## II.D.4 Risk Ranking

A number of ongoing activities in Title III are associated with risk ranking, or have risk ranking as one of their components. Under the Source Category Schedule development program, the schedule for regulation of the listed source categories due to be published for comment this year, has been primarily based on a risk ranking of the various source categories included on the Section 112(c) list. This ranking process uses the Source Category Ranking System (SCRS), a methodology developed within OAQPS. The SCRS process uses health information, available or estimated emissions data, and population data to develop a numerical score for each category on the list. The scores are then ranked to develop a prioritized list. In general, the SCRS first develops a health score for each pollutant emitted by a source category. The health score for each pollutant is based on available data regarding carcinogenicity, reproductive toxicity, acute lethality, and other toxicity. The SCRS then develops an exposure score for each pollutant emitted by that source category. The exposure score is based on concentration approximations for each pollutant from each facility in the category combined with estimates of the numbers of people exposed to these concentration estimates. General assumptions concerning plant stack parameters, plant boundaries, population densities, and meteorological conditions are made on a category-wide basis to simplify the ranking process. Default assumptions and mass balance emission estimates are used where data are unavailable.

The end result of the SCRS process is not an estimate of risk, but rather a score which indicates the relative magnitude of risks between source categories. This score, along with other factors such as efficiency of grouping like sources for a particular regulation, availability of control technology information, and the specific nature of adverse health effects associated with a source category, is then used to assist in the scheduling of regulations.

The Lesser Quantity Emission Rate project is an example of a risk ranking assessment because of the use of exposure assessment and data on health effects. Title III (Section 112(a)) allows the Administrator to establish emission rates for less than 10 and 25 tons/year for HAPs based on their potency, persistence, potential for bioaccumulation, or other relevant factors. The HAPs with UREs and classified as a known, probable, or possible human carcinogen were initially selected. Added to these were chemicals of high concern under CERCLA. Noncarcinogens were selected on the basis of their inhalation RfC, RfD, LC50, or LOEL. Using standard parameters, a generic exposure modeling was done, including consideration of likely exposure duration. This modeling analysis yielded an estimated ambient concentration at a distance selected to represent the nearest residence. This ambient concentration was compared to cancer UREs or noncancer benchmarks, and HAPs of concern were

identified. Lesser quantity emission rates (LQER) were assigned to selected carcinogens based upon order-of-magnitude changes in their potencies. The range of LQERs that resulted was .0001 to 1 ton/year. Selected noncarcinogens were assigned LQERs based on a comparison of the benchmark concentrations with the estimated ambient concentration. The major consequence of this analysis would be a redefinition of some sources as major sources if their emission rates of HAPs exceed the assigned LQER.

## II.D.5 Quantitative Risk Assessment

With regard to quantitative risk assessment activities, two current CAA-related activities address the use of refined modeling techniques with site-specific data to quantify risks associated with both long- and short-term exposure to hazardous air pollutants from stationary sources.

### Source Category Deletion Petition Process

Under Section 112(c), a source category may be deleted from the list of source categories subject to regulation via a petition process if a petition demonstrates, for the case of carcinogenic pollutants, that "no source in the category ... emits (carcinogenic) air pollutants in quantities which may cause a lifetime risk of cancer greater than one in one million to the individual in the population who is most exposed to emissions of such pollutants from the source," and, for the case of noncarcinogenic yet toxic pollutants, that "emissions from no source in the category ... exceed a level which is adequate to protect public health with an ample margin of safety and no adverse environmental effect will result from emissions from any source."

In support of the petition process, EPA is developing guidance for petitioners which suggests acceptable methodologies for assessing cancer and noncancer risk associated with sources of HAPs. This guidance references a document describing a tiered modeling approach for the estimation of maximum risks (see Appendix T - Tiered Modeling Approach). The tiered approach begins with a screening methodology which is used to identify facilities within a source category that do not present risks significant enough to warrant more refined analysis. The screening methodology uses minimal site-specific data (pollutant emission rates, stack heights, and minimum fenceline distances) in this assessment, and, as such, the results are very conservative. Facilities not screened out in this first tier are subjected to a more refined "Tier 2" assessment requiring additional site-specific information (stack diameters, exit velocities, exit temperatures, rural/urban classification, nearest building dimensions) concerning each modeled facility. The third modeling tier requires the most site-specific data (release point and fenceline locations, local meteorological data, release durations and annual frequencies) to provide the most refined estimate of risks due to each modeled source.

The analyses described above focus on the maximum risks presented by a facility outside its plant boundary, regardless of how many people are subjected to those risk levels. To the extent that population location and distribution data are available, they may be incorporated in the analysis on a case-by-case basis, to provide a more accurate estimate of the risk to the maximum exposed individual.

Residual Risk Evaluation

Under Section 112(f) of the CAA, EPA is required to assess the risks associated with a regulated source category within 8 years of the MACT standard promulgated for that category. The Agency is currently evaluating options for implementing this provision. In investigating various alternatives, many questions have been raised. The EPA is currently exploring many technical and policy issues. These issues must be addressed prior to establishing an implementation strategy for evaluating residual risks.

In fully characterizing the potential risks associated with emissions of HAPs following compliance with the MACT standards, EPA is evaluating the capabilities of current risk assessment methods. Presently, due to limited availability of data and methods, it is difficult to quantitatively characterize specific risks (e.g., noncancer risks). The EPA is evaluating various methods to collect additional effects data (see response to Question 4) as well as exploring the development of new methods and the modification of existing methods to improve the ability to quantify risks. Specific areas that are being explored include: evaluation of less-than-lifetime exposures, incorporation of severity-of-effect data, incorporation of data on reversibility (or irreversibility) of effects, and development of physiologically based pharmacokinetic and biologically based dose-response models.

Currently, it is envisioned that a tiered modeling approach (such as described above in the discussion of the source category deletion process) may be the basis for dispersion modeling associated with residual risk analyses of source categories. The EPA envisions that site-specific emission estimates may "drive" the risk assessment process. Thus, EPA is planning its efforts to expand the available emission measurement methods and validation procedures (validated measurement methods currently exist for only about 15-20% of the listed HAPs). In addition, EPA believes that efforts should be extended to continue to improve available emission calculation methods (emission factors, surface impoundment emission estimation methods, etc.). To assist the process of obtaining sufficient site-specific data for quantitative risk analyses, EPA is investigating options for developing a user-access data entry system.

Such a system would necessarily be designed to ease the burden of providing up-to-date data to EPA and to protect against the unauthorized access of proprietary information. Logistics, reporting requirements, and quality assurance associated with such a system are problems with no adequate answers at this point.

The EPA is also looking into improving risk assessments by factoring in more realistic approaches to exposure assessments including consideration of population mobility, population sensitivity, activity patterns, and indoor/outdoor exposures. Because of the intensive data requirements for addressing these factors adequately, sensitivity studies are being considered to assess the ranges of uncertainty induced by each of these factors on the predicted exposures and risks. The results from such studies would hopefully allow a more representative characterization of the distribution of risks among the exposed population in the future.

## II.D.6 Evolution of Exposure and Risk Assessment

As previously mentioned, the role and scope of exposure assessments in the air toxics program is changing. Exposure estimates were conducted for two main purposes: 1) to estimate high end and population exposure to a candidate hazardous air pollutant, and 2) to evaluate the effectiveness of various air pollution control alternatives for reducing potential exposure and risk. Table 3 presents data sources and assumptions that were generally used in previous exposure assessments. The source category deletion and residual risk evaluation provisions in Title III place a much greater focus on source and individual exposures associated with an often complex mixture of source types and pollutants.

Procedures that the Agency develops for addressing residual risk will be designed to meet several criteria. State and local air pollution control agencies, affected industries, and private individuals may require access to and familiarity with available models. In addition, the procedure should be able to evaluate present and future control options as interested parties may wish to evaluate residual risk before air pollution control equipment are ordered.

As noted above, OAQPS is currently examining and developing improved techniques for conducting exposure assessments. Although these improvements will continue to chiefly rely on predictive methods (modeling), measured data, available from monitored levels or reconstructed from measurement of biological fluids and tissues, will remain an important source of information for validation and characterization purposes. The Agency will focus the improvements in three main areas:

1) Developing user-friendly models to enable diverse, interested parties to understand and operate the models if they choose. Data input and selection of specific models will be accomplished by menu screens that contain data checks.

Such a system would necessarily be designed to ease the burden of providing up-to-date data to EPA and to protect against the unauthorized access of proprietary information. Logistics, reporting requirements, and quality assurance associated with such a system are problems with no adequate answers at this point.

The EPA is also looking into improving risk assessments by factoring in more realistic approaches to exposure assessments including consideration of population mobility, population sensitivity, activity patterns, and indoor/outdoor exposures. Because of the intensive data requirements for addressing these factors adequately, sensitivity studies are being considered to assess the ranges of uncertainty induced by each of these factors on the predicted exposures and risks. The results from such studies would hopefully allow a more representative characterization of the distribution of risks among the exposed population in the future.

## II.D.6 Evolution of Exposure and Risk Assessment

As previously mentioned, the role and scope of exposure assessments in the air toxics program is changing. Exposure estimates were conducted for two main purposes: 1) to estimate high end and population exposure to a candidate hazardous air pollutant, and 2) to evaluate the effectiveness of various air pollution control alternatives for reducing potential exposure and risk. Table 3 presents data sources and assumptions that were generally used in previous exposure assessments. The source category deletion and residual risk evaluation provisions in Title III place a much greater focus on source and individual exposures associated with an often complex mixture of source types and pollutants.

Procedures that the Agency develops for addressing residual risk will be designed to meet several criteria. State and local air pollution control agencies, affected industries, and private individuals may require access to and familiarity with available models. In addition, the procedure should be able to evaluate present and future control options as interested parties may wish to evaluate residual risk before air pollution control equipment are ordered.

As noted above, OAQPS is currently examining and developing improved techniques for conducting exposure assessments. Although these improvements will continue to chiefly rely on predictive methods (modeling), measured data, available from monitored levels or reconstructed from measurement of biological fluids and tissues, will remain an important source of information for validation and characterization purposes. The Agency will focus the improvements in three main areas:

1) Developing user-friendly models to enable diverse, interested parties to understand and operate the models if they choose. Data input and selection of specific models will be accomplished by menu screens that contain data checks.

Addition of Monte Carlo techniques to permit the representation of those rameters that greatly affect the exposure/risk estimates by distributions rather an point estimates (see Appendix U, Monte Carlo Approach).

A geographical information system (GIS) will be integrated with the models improve the predicted ambient concentrations by incorporation of topography d land use information to aid in selection of appropriate meteorological data d the location of area source categories. In addition, GIS will allow OAQPS more accurately locate areas where people may reside than is currently ssible using U.S. Bureau of Census data alone (See Appendix V, GIS - pplication to Exposure Assessment).

T f input parameters that can presently be described by distributions include:

emission rates
unit risk factor (carcinogenic potency estimate)
microenvironment concentrations
time spent in each microenvironment
information on the length of time people are expected to reside in their primary residences
the ability to vary the location of the predicted ambient concentrations.

e EPA/OAQPS is also developing a separate model (Hazardous Air Pollutant
E. Model (HAPEM)) examining the impact on exposure of population mobility
(e muting) (see Appendix W, HAPEM - Mobility Considerations).

ce the process of conducting residual risk analyses for all regulated sources
of s anticipated to be a resource-intensive process, the analytical methodology
h: d into a tiered approach, as mentioned above. This differs from most risk
a: ts performed in the past in that it allows for the incorporation of site-specific
d: e possible to refine the estimates of population exposure and risk. Since it
w be difficult for EPA to require all regulated facilities to provide all of the
n: data for such site-specific analyses, EPA has plans to develop a voluntary
d: ge and retrieval system, whereby such facilities may provide site-specific
d: PA to facilitate the more rigorous risk assessment process. This will not only
he to perform residual risk analyses in a more efficient manner, but it will
re: level of "unnecessary" conservatism associated with the risk assessment
pr In situations where EPA does not have site-specific modeling parameters, the
ris sments will be performed at the Tier 1 level, consistent with risk assessments
of t. In situations where additional data have been provided

by ilities being analyzed, risk assessments will be more realistic, and risk
est will generally be lower (sometimes by orders of magnitude).

Table 4 below summarizes the major differences between the 3 modeling tiers discussed above by briefly listing the input requirements, the major output parameters, and the assumptions associated with each tier. This table may be used to quickly determine whether a given scenario may be modeled at any particular tier based on available site-specific data. Within each tier, cancer unit risk estimates, chronic noncancer concentration thresholds, and acute concentration thresholds are required to convert concentration predictions into cancer risks, chronic noncancer risks, and acute noncancer risks, respectively.

Table 4: Exposure Modeling Parameters

| Modeling Tier | Input Requirements | Output Parameters | Major Assumptions |
|---|---|---|---|
| Tier 1 | emission rate, stack height, minimum distance to fenceline | maximum off-site concentrations, worst-case cancer risk or worst-case noncancer hazard index (short- and long-term) | Worst-case meteorology, worst-case downwash, worst-case stack parameters, short-term releases occur simultaneously, maximum impacts co-located, cancer risks additive, noncancer risks additive |
| Tier 2 | emission rate, stack height, minimum distance to fenceline, stack velocity, stack temperature, stack diameter, rural/urban site classification, building dimensions for downwash calculation | maximum offsite concentrations, worst-case cancer risk and/or worst-case noncancer hazard index (short- and long-term) | Worst-case meteorology, short-term releases occur simultaneously, maximum impacts co-located, cancer risks additive, noncancer risks additive |
| Tier 3 | emission rate, stack height, actual fenceline and release point locations, stack velocity, stack temperature, stack diameter, rural/urban site classification, local meteorological data, receptor locations for concentration predictions, frequency and duration of short-term (intermittent) releases | concentrations at each receptor point, long-term cancer risk estimates, chronic noncancer hazard index estimates at each receptor point, annual hazard index exceedance rate at each receptor | cancer risks additive, noncancer risks additive |

In general, to perform a site-specific exposure assessment, Tiers 1 and 2 could be used to screen facilities with low risk estimates from further analysis at a higher

Tier. In cases where facility-specific data are lacking, emissions estimates could be made using a model plant approach with emission factors or process engineering estimates of emissions. In such cases, all known or estimated emissions could be assumed to emanate from a single, typical stack at the plant center, and the plant could be assumed to have a circular boundary, 200 meters from the plant center. It is anticipated that plant location data (latitude and longitude) will be obtained from EPA permits, and this would allow predicted ambient concentration levels to be compared to potentially-exposed populations through the use of U.S. Census Bureau data. It is also anticipated that more rigorous analyses to provide the distribution of risks among exposed population would be performed where site data are sufficient to support such analyses.

The major influence of the guidelines on exposure assessments is in the quantification of uncertainty. The HEM is being redesigned to explicitly address uncertainty quantitatively where possible. A discussion of risk characterization and attempts to describe and communicate uncertainty was presented previously in Section II.B.4.

## III. Question 3 What HAPs data are available now to implement the current risk assessment methodology?

### III.A Introduction

The EPA has compiled currently available data on the hazardous air pollutants (HAPs) in developing strategies for implementing various provisions contained in Title III of the Clean Air Act. These data include: information on the schedule for control technology-based standards, recent annual air emissions data, preliminary estimates of the number of facilities that emit HAPs, and health effects information.

### III.B Summaries of Available Data

Table 5 is an summary of the currently available health data (this is an updated version of the Table previously provided), taken from Table 6.

and by cooperative agreements with non-profit organizations (e.g., universiti
A critical provision of the RIHRA program is that it includes a competitive process for selection of individual research efforts. As projects come to completion their funds are returned to a central pool from which research renewals and new projects are funded. Extramural review of project proposals and of the RIHRA research strategy occurs as part of this process, and we have been pleased by the very positive support voiced by reviewers regarding the program.

In addition to the RIHRA program there are complementary health research activities underway to address the specific needs of EPA regulatory programs (e.g., air, water). I believe it is important that you be aware of these programs and their relationship to risk assessment. The very strong relationship of these research activities to the risk assessment paradigm is presented in several documents enclosed for your information. The document "Strategy for Environmental Health Research at EPA" presents our strategic plan for conducting health research that is responsive to the regulatory needs of the EPA, but is also consistent with the underlying scientific issues associated with the risk assessment process. This document presents a very strong conceptual framework and strategic plan for EPA's health research programs. The document "The Role of Health Research in Support of EPA's Regulatory Programs" documents EPA's regulatory mandates and presents the linkage between health research activities and regulatory program needs. The conceptual framework for conducting health research relevant to the EPA's regulatory programs is again closely aligned in this document with the risk assessment paradigm. The briefing package "Congressional Staff Visit" provides ar overview of our research strategy and presents examples of research accomplishments. Please let me know if questions arise regarding these other research programs and I will provide you with additional information or conta

I, or Dr. John Vandenberg, RIHRA Director, would be pleased to discuss any aspect of our research activities with you and to address any questions you may have regarding the enclosed materials. We also would be pleased to provide a brief presentation on our research program to your committee. Please contact me (919-541-2281) or John (919-541-4527) if we can be of assistance.

Sincerely,

Lawrence W Reiter, Ph.D.
Director

Enclosures

cc: William Farland
Ken Sexton
John Vandenberg

nasletter.hap

Table 5: Summary of Health Effects Data (December 10, 1992)

| Status | Cancer | Noncancer |
|---|---|---|
| Verified RfC<br>On IRIS<br>Not on IRIS | | <br>30<br>5 |
| Reviewed, not verifiable | | 53 |
| WOE and IUR | 40 | |
| WOE and OUR | 14 | |
| WOE Only | 34 | |
| Under review[2] | 10 | 25 |
| No status | 88 | 73 |
| Total HAPs | 186 | 186 |

[1] Does not include lead, radionuclides, or glycol ethers

[2] Under review by Environmental Criteria and Assessment Office or Human Health Assessment Group for derivation of RfC or URE followed by verification review by RfC/RfD and CRAVE work groups before entering data onto IRIS

RfC: Inhalation reference concentration
WOE: Weight-of-evidence, includes A to D class.
IUR: Inhalation unit risk estimate
OUR: Oral unit risk estimate

**CURRENT DATA ON THE HAPs**
**Dec. 10,1992**

| CAS # | Chemical Name | | MACT YEAR 4 | MACT YEAR 7 | MACT YEAR 10 | 1990 Emis (T/yr) | 1989 Emis (T/yr) | 1988 Emis (T/yr) | # of Emit Facil | IUR per ug/m3 | OUR per ug/L | EPA WOE | RfC mg/m3 Stat | IARC WOE | Exp. Asses. | Gene MVV Sc | Gene MVV G | Tox MVT M | Tox MVT C | Data NM S | Data NM E | Repro/ Dev Data |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Column Number | | | 1 | | 2 | 2 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | | 12 | | 13 | | 14 |
| | | #SC | | | | | | | | | | | | | | | | | | | | |

Column Numbers and Footnotes to Table

1 Chemicals emitted from sources that will be regulated within the 2, 4, 7, and 10 year deadlines for maximum achievable control technology standards (# preceding X indicates the # of source categories (#SC) HAP in).

2 and 3 Toxic Release Inventory Data (TRI) in tons/year (1988,1989,1990)

4 Number of facilities reporting emissions to TRI in 1990

5 IUR= Inhalation unit risk estimate per ug/m3; Source is EPA's Integrated Risk Information System (IRIS)

6 OUR= Oral unit risk estimate per ug/l; Source is EPA's IRIS data base

7 WOE= Weight of Evidence classification; Source is EPA's IRIS data base

8 RfC workgroup; verified, on IRIS= conc. given in mg/m3
verified, not on IRIS= 'v'

9 IARC (International Agency for Research on Cancer) WOE

10 Exposure Assessments:
A) HAPS with risk assessments done for development of Section 112 standards
B) HAPS with screening assessments done for listing purposes

11, 12, 13 Information on Genetic Toxicology; Source= Genetic Activity Profiles data base provided by Dr. Michael Waters, EPA's Health Effects Research Lab.
The + or - represents the overall call for that group which may contain
more than one assay. When discrepancies exist within a group, this is indicated by a +- (or-+).
The first symbol represents the majority call for that group.

CURRENT DATA ON THE HAPs
Dec. 10,1992

| CAS # | Chemical Name | MACT 1990 Emis (T/yr) 1 | 1989 Emis (T/yr) 2 | 1988 Emis (T/yr) 3 | # of Facil 4 | IUR per ug/m3 5 | OUR per ug/L 6 | EPA WOE 7 | HO mg/m3 Stat 8 | LARG WOE 9 | Rep. Assess. 10 | Gene MVV So G 11 | Ter MVT M C 12 | Data NM S E 13 | Repr/ Dev Data 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Column Number | | | | | | | | | | | | | | | |
| | #60 | | | | | | | | | | | | | | |

MVV= Mammalian, In Vivo
So= Somatic cell; G= Germ cell
MVT= Mammalian, In Vitro
M= Mutation; C= Chromosome aberration
NM= Non-mammalian
S= Salmonella typhi.; E= Escherichia coli

14 Data from Non-cancer Health Effects Database, prepared and provided by Dr. John Vandenberg, EPA's Health Effects Research Lab.
'X' indicates data available; X* indicates some human data available
Note: Data includes effects on maternal toxicity: All data from inhalation exposure

**CURRENT DATA ON THE HAPs**
**Dec. 10,1992**

| CAS # | Chemical Name | MACT YEAR 24 7 10 | 1990 Emis (T/yr) | 1989 Emis (T/yr) | 1988 Emis (T/yr) | # of Emit Facil | IUR per ug/m3 | OUR per ug/L | EPA WOE | HC mg/m3 Stat | IARC WOE | Exp. Assess. | Gene MVV Se | Gene MVV C | Tox MVT M | Tox MVT C | Data NM S | Data NM E | Report Dev Data |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Column Number | | 1 | 2 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | | 12 | | 13 | | 14 |
| | #SC | | | | | | | | | | | | | | | | | | |

Symbols used: UR= under review
V= verified
NA= not available
NV= not verifiable

IARC vs. EPA: Classification Differences

EPA Modifications to IARC Approach:
1. Considers statistically significant association between an agent and life-threatening benign tumors when evaluation human risk
2. Added "no data available" category
3. Added "no evidence of carcinogenicity" category

By Category

EPA
Group A - Known human carcinogen
Group B - Probable human carcinogen
B1 - Limited human data
B2 - Inadequate human data, sufficient animal data
Group C - Possible human carcinogen
- No human data, limited animal data
Group D - Not classifiable as to human carcinogenicity
- Inadequate or no human or animal data
Group E - Evidence of noncarcinogenicity for humans
- No evidence in at least 2 adequate animal tests

IARC
Group 1 - Known human carcinogen
Group 2A - Probable human carcinogen
Group 2B - Possible human carcinogen
Group 3 - Not classifiable as to human carcinogenicity
Group 4 - Probably not carcinogenic to humans

**CURRENT DATA ON THE HAPs**
**Dec. 10,1992**

| CAS # | Chemical Name | | MACT 1990 Emis (T/yr) | 1990 Emis (T/yr) | 1969 Emis (T/yr) | 1968 Emis (T/yr) | # of Emiss Facil | IUR per ug/m3 | OUR per ug/L | EPA WOE | RfC mg/m3 Stat | IARC WOE | Exp. Assoc. | Gene MVV So | Gene MVV G | Tox MVT M | Tox MVT C | Data NM S | Data NM E | Repro/ Dev Data |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Column Number | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | | | | |
| | | #60 | | | | | | | | | | | | | | | | | | |

MVV= Mammalian, In Vivo
So= Somatic cell; G= Germ cell
MVT= Mammalian, In Vitro
M= Mutation; C= Chromosome aberration
NM= Non-mammalian
S= Salmonella typhi.; E= Escherichia coli

14 Data from Non-cancer Health Effects Database, prepared and provided by Dr. John Vandenberg, EPA's Health Effects Research Lab.
'X' indicates data available; X* indicates some human data available
Note: Data includes effects on maternal toxicity: All data from Inhalation exposure

**CURRENT DATA ON THE HAPs**
**Dec. 10,1992**

| CAS # | Chemical Name | MACT YEAR 2 4 7 10 | 1990 Emis (T/yr) | 1989 Emis (T/yr) | 1988 Emis (T/yr) | # of Emit Facil | IUR per ug/m3 | OUR per ug/L | EPA WOE | MU mg/m3 Stat | IARC WOE | Exp. Assoc. | Gene MVV Sc | G | Tox MVT M | G | Data MM S | E | Repro/ Dev Data |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Column Number | 1 | 2 | 3 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | | 12 | | 13 | | 14 |
| | SIC | | | | | | | | | | | | | | | | | | |

Symbols used: UR= under review
V= verified
NA= not available
NV= not verifiable

IARC vs. EPA: Classification Differences

EPA Modifications to IARC Approach:
1. Considers statistically significant association between an agent and life-threatening benign tumors when evaluation human risk
2. Added "no data available" category
3. Added "no evidence of carcinogenicity" category

By Category

EPA

Group A - Known human carcinogen
Group B - Probable human carcinogen
B1 - Limited human data
B2 - Inadequate human data, sufficient animal data
Group C - Possible human carcinogen
- No human data, limited animal data
Group D - Not classifiable as to human carcinogenicity
- Inadequate or no human or animal data
Group E - Evidence of noncarcinogenicity for humans
- No evidence in at least 2 adequate animal tests

IARC

Group 1 - Known human carcinogen
Group 2A - Probable human carcinogen
Group 2B - Possible human carcinogen
Group 3 - Not classifiable as to human carcinogenicity
Group 4 - Probably not carcinogenic to humans

CURRENT DATA ON THE HAPs
Dec. 10,1992

| CAS # | Chemical Name | MY 2 | AE 4 | CA 7 | TR 10 | 1990 Emis (T/yr) 1 | 1989 Emis (T/yr) 2 | 1988 Emis (T/yr) 3 | # of Facil 4 | IUR per ug/m3 5 | OUR per ug/L 6 | EPA WOE 7 | RfC mg/m3 Stat 8 | IARC WOE 9 | Exp. Assoc. 10 | Gene MVV Se 11 | Gene MVV C 11 | Tox MVT M 12 | Tox MVT C 12 | Data NM S 13 | Data NM E 13 | Repro/ Dev Data 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Column Number | | | 1 | | | | | | | | | | | | | | | | | | |
| | #SO | | | | | | | | | | | | | | | | | | | | | |
| 79345 | 1,1,2,2-Tetrachloroethane | 2 | X | | X | 22.3 | 17.7 | 22.9 | 20 | 5.8E-05 | 5.8E-06 | C | | 3 | | | | | | + | | X |
| 79005 | 1,1,2-Trichloroethane | 2 | X | | X | 299.4 | 398.4 | 870.1 | 28 | 1.6E-05 | 1.6E-06 | C | NV | 3 | | | | | | - | | |
| 57147 | 1,1-Dimethyl hydrazine | 1 | X | | | 0.2 | 0.4 | 2.2 | 4 | | | UR | NV | 2B | | | | | | | | |
| 120821 | 1,2,4-Trichlorobenzene | 1 | X | | | 168.4 | 575.7 | 760.1 | 61 | | | D | | | | + | | | | - | | X |
| 96128 | 1,2-Dibromo-3-chloropropane | | | | | | NA | | | | 4.0E-05 | B2 | 2.0E-04 | 2B | | | -+ | | + | + | + | X* |
| 122667 | 1,2-Diphenylhydrazine | | | | | | NA | | | 2.2E-04 | 2.2E-05 | B2 | NV | | | | | | | + | - | |
| 106887 | 1,2-Epoxybutane (1,2-Butylene oxide) | | | | | 39.7 | 59.8 | 54.0 | 21 | | | | 2.0E-02 | | | | | + | + | + | | |
| 75558 | 1,2-Propylenimine (2-Methyl aziridine) | | | | | 0.3 | 0.3 | 0.3 | 6 | | | | NV | 2B | | | | | | | | |
| 106990 | 1,3-Butadiene | 18 | | X | X | X | 2518.8 | 2768.7 | 3268.4 | 161 | 2.8E-04 | | B2 | | 2B | B | + | | | + | | X |
| 542756 | 1,3-Dichloropropene | 1 | X | | | | 29.7 | 25.5 | 28.2 | 7 | | | | 2.0E-02 | 2B | | | | | + | | X |
| 1120714 | 1,3-Propane sultone | | | | | | | NA | | | | | | | 2B | | | | | | | |
| 106467 | 1,4-Dichlorobenzene(p) | 1 | X | | | | 409.1 | 793.6 | 904.2 | 28 | | | V | | 2B | | | | | - | | |
| 123911 | 1,4-Dioxane (1,4-Diethyleneoxide) | 1 | X | | | | 299.2 | 390.0 | 270.4 | 88 | | 3.1E-07 | B2 | | 1B | | | | - | - | | |
| 540841 | 2,2,4-Trimethylpentane | 3 | X | X | X | | | NA | | | | | | NV | | | | | | | | |
| 1746016 | 2,3,7,8-Tetrachlorodibenzo-p-dioxin | | | | | | | NA | | | | | | | 2B | | - | | + | - | | X* |
| 95954 | 2,4,5-Trichlorophenol | 1 | X | | | | | 0.1 | 0.0 | | | | | NV | | | | | | - | | |
| 88062 | 2,4,6-Trichlorophenol | | | | | | 0.0 | 0.1 | 0.1 | 1 | 3.1E-06 | 3.1E-07 | B2 | NV | 2B | | | | + | - | | |
| 94757 | 2,4-D, salts,esters(Dichlorophenoxyacetic a | 1 | | | | X | 3.9 | 3.6 | 3.5 | 27 | | | | | 2B | | - | - | + | + | | X* |
| 51285 | 2,4-Dinitrophenol | 1 | X | | | | 12.3 | 6.8 | 10.4 | 11 | | | | NV | | | | | | - | + | |
| 121142 | 2,4-Dinitrotoluene | 1 | X | | | | 28.8 | 43.6 | 46.1 | 11 | | 1.9E-05 | B2 | NV | | | - | - | -+ | + | - | X* |
| 95807 | 2,4-Toluene diamine | 1 | X | | | | 2.0 | 2.2 | 1.5 | 5 | | | | NV | 2B | | | | | | | |
| 584849 | 2,4-Toluene diisocyanate | 2 | X | X | | | 28.7 | 61.3 | 113.9 | 113 | | | | V | 2B | | | | | - | | |
| 53963 | 2-Acetylaminofluorene | | | | | | | NA | | | | | | | | | | | | + | + | |
| 532274 | 2-Chloroacetophenone | 1 | X | | | | | NA | | | | | | 3.0E-05 | | | | | | | | |
| 79469 | 2-Nitropropane | 1 | X | | | | 42.1 | 112.6 | 418.6 | 8 | | | | 2.0E-02 | 2B | | | | | | | |
| 119904 | 3,3'-Dimethoxybenzidine | | | | | | 0.0 | 0.3 | | 4 | | | | | 2B | | | | | +. | | |
| 119937 | 3,3'-Dimethyl benzidine | 1 | X | | | | | NA | | | | | | NV | 2B | | | | | | | |
| 91941 | 3,3-Dichlorobenzidine | | | | | | 0.0 | 0.1 | 0.1 | 7 | | 1.3E-05 | B2 | NV | 2B | | | | | + | | |
| 101779 | 4,4'-Methylenedianiline | 1 | X | | | | 9.8 | 23.9 | 75.1 | 29 | | | | UR | 2B | | | | | + | | |
| 101144 | 4,4-Methylene bis(2-chloroaniline) | 1 | X | | | | 1.4 | 0.6 | 0.4 | 16 | | | | | 2A | | | | | | | |
| 534521 | 4,6-Dinitro-o-cresol, and salts | | | | | | 0.0 | 0.1 | | 10 | | | | | | | | | | | | |
| 92671 | 4-Aminobiphenyl | | | | | | | 0.0 | | | | | | | 1 | | | + | + | + | + | |
| 92933 | 4-Nitrobiphenyl | | | | | | | NA | | | | | | | 3 | | | | | | | |

**CURRENT DATA ON THE HAPs**
**Dec. 10,1992**

| CAS # | Chemical Name | Column Number | MY R | AE 4 | CA 7 1 | TR 10 | 1990 Emis (T/yr) 1 | 1989 Emis (T/yr) 2 | 1968 Emis (T/yr) 3 | # of Emit Facil 4 | IUR per ug/m3 5 | OUR per ug/L 6 | EPA WOE 7 | RfC mg/m3 Stat 8 | IARC WOE 9 | Exp. Assoc. 10 | Gene MVV Se 11 | Gene MVV G 11 | Tox MVT M 12 | Tox MVT C 12 | Data NM S 13 | Data NM R 13 | Repro/ Dev Data 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | #80 | | | | | | | | | | | | | | | | | | | | | |
| 100027 | 4-Nitrophenol | 1 | X | | | | 3.6 | 3.9 | 3.9 | 6 | | | | NV | | | | | | | | | X |
| 75070 | Acetaldehyde | 7 | | X | X | X | 8440.3 | 8762.1 | 8336.6 | 77 | 2.2E-06 | | B2 | 9.0E-03 | 2B | | | + | | + | + | + | X |
| 60355 | Acetamide | 1 | X | | | | 0.0 | NA | | 3 | | | | | 2B | | | | | | | | |
| 75058 | Acetonitrile | 1 | X | | | | 881.8 | 693.4 | 1022.0 | 73 | | | | UR | | | | | | | | | X |
| 98862 | Acetophenone | 1 | X | | | | | NA | | | | | D | NV | | | | | | | | | |
| 107028 | Acrolein | 7 | X | | X | | 11.0 | 2.2 | 16.6 | 16 | | | C | 2.0E-05 | 3 | | | - | | + | + | | X |
| 79061 | Acrylamide | 2 | X | | X | | 25.0 | 12.5 | 13.0 | 69 | 1.3E-03 | 1.3E-04 | B2 | NV | 2B | | - | + | | | - | | |
| 79107 | Acrylic acid | 2 | X | X | | | 213.5 | 178.7 | 399.3 | 183 | | | | 3.0E-04 | 3 | | | | | | | | X |
| 107131 | Acrylonitrile | 7 | X | X | X | X | 1574.0 | 2191.9 | 2111.7 | 120 | 6.8E-05 | 1.5E-05 | B1 | 2.0E-03 | 2A | B | - | - | + | +- | + | + | X |
| 107051 | Allyl chloride | 1 | X | | | | 103.0 | 87.8 | 73.2 | 21 | | | C | 1.0E-03 | 3 | | | | | | + | | X |
| 62533 | Aniline | 2 | X | | X | | 237.4 | 252.1 | 257.2 | 72 | | 1.6E-07 | B2 | 1.0E-03 | 3 | | - | | + | + | - | - | |
| 0 | Antimony Compounds | 3 | | | X | X | 73.0 | 79.3 | 78.8 | 418 | | | | | | | | | | | | | |
| 0 | Arsenic Compounds (inorganic including ar | 11 | | | X | X | 82.9 | 87.9 | 156.7 | 347 | 4.3E-03 | | A | UR | 1 | A | + | - | - | + | - | - | |
| 1332214 | Asbestos | | A* | | | | 8.7 | 18.7 | 23.8 | 99 | 2.2E-01 | Fib/ML | A | | 1 | A | - | | -+ | +- | - | - | |
| 71432 | Benzene | 27 | | X | X | X | 12203.4 | 12341.5 | 14144.9 | 495 | 8.3E-06 | 8.3E-07 | A | UR | 1 | A | +- | | -+ | - | - | - | X* |
| 92875 | Benzidine | 1 | | | X | | | NA | | | 6.7E-02 | 6.7E-03 | A | NV | 1 | | +- | | +- | + | + | - | |
| 98077 | Benzotrichloride | 1 | X | | | | 4.2 | 12.6 | 12.5 | 4 | | 3.6E-04 | B2 | | | | | | | | + | + | |
| 100447 | Benzyl chloride | 3 | X | | X | X | 16.8 | 13.6 | 21.7 | 51 | | 4.9E-06 | B2 | NV | | | - | | +- | +- | + | + | |
| 0 | Beryllium Compounds | 3 | | | | X | 0.1 | 1.7 | 2.3 | 3 | 2.4E-03 | 1.2E-04 | B2 | UR | 2A | A | | | + | + | - | + | |
| 57578 | beta-Propiolactone | 2 | X | | X | | | NA | | | | | | NV | 2B | | | | | | | | |
| 92524 | Biphenyl | 3 | X | | X | X | 560.5 | 544.1 | 604.3 | 152 | | | D | NV | | | | | | | | | |
| 111444 | Bis(2-chloroethyl)ether (Dichloroethyl ethe | 1 | X | | | | 1.9 | 2.4 | 2.5 | 11 | 3.3E-04 | 3.3E-05 | B2 | NV | 3 | | | - | | | + | | X |
| 117817 | Bis(2-ethylhexyl)phthalate (DEHP) | | | | | | 672.3 | 539.4 | 563.9 | 349 | | 4.0E-07 | B2 | | 2 | | - | + | - | - | - | - | X |
| 542881 | Bis(chloromethyl)ether | 1 | X | | | | 0.0 | 0.0 | 0.0 | 3 | 6.2E-02 | 6.2E-03 | A | NV | 1 | | | | | | + | | X |
| 75252 | Bromoform | 1 | X | | | | 24.1 | NA | | 3 | 1.1E-06 | 2.3E-07 | B2 | | | | | | | - | - | | |
| 0 | Cadmium Compounds | 16 | | X | X | X | 45.0 | 59.9 | 64.8 | 159 | 1.8E-03 | | B1 | UR | 2A | B | - | - | + | +- | | + | |
| 156627 | Calcium cyanamide | | | | | | 6.3 | 6.3 | 6.3 | 3 | | | | | | | | | | | | | |
| 105602 | Caprolactam | 4 | X | | X | X | | NA | | | | | | | 4 | | | | - | -+ | - | | X |
| 133062 | Captan | | | | | | 9.6 | 12.6 | 7.9 | 20 | | | UR | NV | 3 | | | | | | | | |
| 63252 | Carbaryl | 1 | X | | | | 4.6 | 5.0 | 3.7 | 27 | | | | NV | 3 | | | | | | | | |
| 75150 | Carbon disulfide | 4 | | | X | X | 49111.3 | 49897.7 | 62590.2 | 85 | | | | UR | | | + | + | | | - | | X* |
| 56235 | Carbon tetrachloride | 11 | X | X | X | X | 835.5 | 1683.6 | 1882.2 | 97 | 1.5E-05 | 3.7E-06 | B2 | | 2B | B | | - | | | - | + | X |
| 463581 | Carbonyl sulfide | 3 | | | X | X | 9317.4 | 9842.5 | 8994.6 | 39 | | | | NV | | | | | | | | | |

**CURRENT DATA ON THE HAPs**
**Dec. 10,1992**

| CAS # | Chemical Name | Column Number | | M Y 2 | A E 4 | O A 7 1 | T R 10 | 1990 Emis (T/yr) 2 | 1989 Emis (T/yr) 2 | 1988 Emis (T/yr) 3 | # of Emit Facil 4 | IUR per ug/m3 5 | OUR per ug/L 6 | EPA WOE 7 | RfC mg/m3 Stat 8 | IARC WOE 9 | Exp. Asses. 10 | Gene MVV Se | Gene MVV G 11 | Tox MVT M | Tox MVT C 12 | Data Mil S | Data Mil E 13 | Rep/ Dev Data 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 120809 | Catechol | | | | | | | 13.9 | 2.0 | 1.6 | 131 | | | | | 3 | | | | | | | | |
| 133904 | Chloramben | | | | | | | 0.0 | NA | | 1 | | | | | | | | | | | | | |
| 57749 | Chlordane | | | | | | | 2.2 | 1.9 | 0.3 | 2 | 3.7E-04 | 3.7E-05 | B2 | UR | 3 | | | - | -+ | | - | | |
| 7782505 | Chlorine | 11 | | | X | X | | 52458.9 | 66174.1 | 66741.7 | 1786 | | | | UR | | | | | | | | | |
| 79118 | Chloroacetic acid | 3 | X | | X | X | | 12.7 | 13.4 | 18.0 | 36 | | | | | | | | | | | | | |
| 108907 | Chlorobenzene | 3 | X | | X | X | | 2023.4 | 2025.4 | 1965.6 | 70 | | | D | UR | | B | + | | + | - | - | | X |
| 510156 | Chlorobensilate | | | | | | | | NA | | | | | | | 2 | | | | | | | | |
| 67663 | Chloroform | 6 | X | X | X | X | | 10881.2 | 12134.0 | 11265.0 | 188 | 2.3E-05 | 1.7E-07 | B2 | UR | 2B | B | - | | - | - | - | - | X |
| 107302 | Chloromethyl methyl ether | | | | | | | 0.1 | 0.1 | 0.1 | 4 | | | A | NV | 1 | | | | | | + | | |
| 126998 | Chloroprene (2-chloro-1,3-butadiene) | 2 | X | X | | | | 87.6 | 503.2 | 609.1 | 9 | | | | V | 3 | B | + | + | - | | + | | X* |
| 0 | Chromium Compounds (+6 FOR IRIS) | 31 | | | X | X | X | 384.8 | 1119.2 | 603.9 | 1484 | 1.2E-02 | | A | UR | 1 | B | + | + | + | + | + | + | |
| 0 | Cobalt Compounds | 3 | | | | X | | 25.9 | 60.8 | 43.0 | 204 | | | | | | | | | | | + | | |
| 0 | Coke Oven Emissions | 2 | | | X | | X | | NA | | | 6.2E-04 | | A | | 1 | A | | | | | | | |
| 1319773 | Cresols/Cresylic acid (isomers and mixture) | 4 | X | | | X | X | 366.3 | 446.9 | 333.6 | 131 | | | | NV | | | | | | | | | |
| 98828 | Cumene (isopropylbenzene) | 3 | X | X | | | X | 2051.6 | 2197.5 | 2359.0 | 181 | | | | UR | | | | | | | | | |
| 0 | Cyanide Compounds | 6 | | | X | X | | 569.1 | 274.5 | 317.5 | 315 | | | D | | | | | | | | | | |
| 72559 | DDE (p,p'-Dichlorodiphenyldichloroethylene) | | | | | | | | NA | | | | 9.7E-06 | B2 | | | | | | + | + | - | - | |
| 334883 | Diazomethane | | | | | | | | NA | | | | | | NV | 3 | | | | | | | | |
| 132649 | Dibenzofuran | | | | | | | 15.1 | 31.9 | 35.4 | 54 | | | D | NV | | | | | | | | | |
| 84742 | Dibutylphthalate | 3 | | | X | X | X | 54.1 | 116.1 | 107.8 | 148 | | | D | NV | | | | | | + | + | | X |
| 62737 | Dichlorvos | | | | | | | 0.7 | 0.7 | 0.5 | 11 | | 5.7E-06 | C | 5.0E-04 | 3 | | | | | | | | X |
| 111422 | Diethanolamine | 1 | X | | | | | 191.8 | 242.1 | 314.1 | 372 | | | | | | | | | | | | | |
| 64675 | Diethyl sulfate | | | | | | | 2.7 | 4.4 | 3.0 | 36 | | | | NV | 2A | | | + | + | + | + | + | |
| 60117 | Dimethyl aminoazobenzene | | | | | | | | NA | | | | | | | | | | | | | | | |
| 79447 | Dimethyl carbamoyl chloride | | | | | | | | NA | | | | | | | 2A | | + | | + | -+ | + | | |
| 68122 | Dimethyl formamide | 2 | X | | | | X | | NA | | | | | | 3.0E-02 | | | - | | -+ | - | - | | X |
| 131113 | Dimethyl phthalate | 2 | X | | | | X | 166.8 | 181.7 | 110.8 | 61 | | | D | NV | | | | | | - | - | | |
| 77781 | Dimethyl sulfate | 1 | X | | | | | 4.9 | 8.2 | 5.4 | 31 | | | B2 | NV | 2A | | + | + | + | + | + | + | |
| 106898 | Epichlorohydrin (1-chloro-2,3-epoxypropan | 4 | X | X | | | | 213.7 | 234.0 | 195.0 | 89 | 1.2E-06 | 2.8E-07 | B2 | 1.0E-03 | 2A | B | -+ | - | +- | + | + | + | X |
| 140885 | Ethyl acrylate | 3 | X | X | X | | | 102.1 | 85.6 | 126.4 | 102 | | | UR | | 2B | | + | | | + | - | | X |
| 100414 | Ethyl benzene | 23 | X | X | X | X | | 4308.8 | 4270.2 | 3358.5 | 775 | | | D | 1.0E+00 | | | - | | + | | - | | X |
| 51796 | Ethyl carbamate (Urethane) | | | | | | | 2.0 | 1.7 | 72.7 | 12 | | | | NV | 2B | | | | | | | | |
| 75003 | Ethyl chloride (Chloroethane) | 4 | X | | | X | X | 1971.0 | 2394.1 | 2310.5 | 48 | | | | 1.0E+01 | | | | | | | | | X |

**CURRENT DATA ON THE HAPs**
**Dec. 10,1992**

| CAS # | Chemical Name | MY 93 | AE 4 | CA 7 | TR 10 | 1990 Emis (T/yr) | 1989 Emis (T/yr) | 1988 Emis (T/yr) | # of Facil | IUR per ug/m3 | OUR per ug/L | EPA WOE | RfC mg/m3 Stat | IARC WOE | Exp. Asses. | Cancer Sc | Cancer MVV C | Tox MVT M | Tox MVT O | Data NM S | Data NM E | Report Dev Data |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Column Number | 1 | | | 1 | 2 | 3 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | | 12 | | 13 | | 14 |
| | #60 | | | | | | | | | | | | | | | | | | | | | |
| 106934 | Ethylene dibromide (Dibromoethane) | 1 | X | | | 29.0 | 29.6 | 31.7 | 32 | 2.2E-04 | 2.5E-03 | B2 | 2.0E-04 | 2A | | - | - | + | + | + | + | X* |
| 107062 | Ethylene dichloride (1,2-Dichloroethane) | 4 | X | X | X | X | 2798.0 | 2955.0 | 2383.7 | 109 | 2.6E-05 | 2.6E-06 | B2 | | 2B | B | - | - | + | | + | - | X |
| 107211 | Ethylene glycol | 3 | X | X | | | 4694.4 | 6446.1 | 6640.3 | 1465 | | | | | | | | | | | | | |
| 151564 | Ethylene imine (Aziridine) | | | | | | | NA | 0.3 | | | | | NV | 3 | | | | | | | | |
| 75218 | Ethylene oxide | 4 | X | X | X | | 1223.7 | 1514.5 | 2300.0 | 184 | | | | | 2A | | + | + | + | + | + | + | X |
| 96457 | Ethylene thiourea | | | | | | 0.0 | 0.4 | 0.3 | 8 | | | | | 2B | B | - | - | - | - | + | + | |
| 75343 | Ethylidene dichloride (1,1-Dichloroethane) | 5 | | | X | X | | NA | | | | | C | UR | | | | | | | | | |
| 0 | Fine mineral fibers | 1 | | | | X | | | | | | | | | | | | | | | | | |
| 50000 | Formaldehyde | 22 | X | X | X | X | 6383.0 | 6281.0 | 6199.7 | 861 | 1.3E-05 | | B1 | | 2A | | -+ | - | + | + | + | + | X* |
| 0 | Glycol ethers | 2 | X | X | | | | NA | | | | | | | | | | | | | | | |
| 76448 | Heptachlor | | | | | | 0.4 | 1.7 | 24.5 | 2 | 1.3E-03 | 1.3E-04 | B2 | | | | | - | - | | + | | |
| 118741 | Hexachlorobenzene | 1 | X | | | | 0.7 | 2.3 | 2.5 | 8 | 4.6E-04 | 4.6E-05 | B2 | NV | 2B | | | - | | - | - | | |
| 87683 | Hexachlorobutadiene | 1 | X | | | | 2.5 | 1.8 | 1.3 | 8 | 2.2E-05 | 2.2E-06 | C | | 3 | | | | | - | - | | X |
| 77474 | Hexachlorocyclopentadiene | | | | | | 42.3 | 44.6 | 7.4 | 4 | | | D | | | B | | | | | | | |
| 67721 | Hexachloroethane | 1 | X | | | | 4.0 | 8.5 | 9.6 | 17 | 4.0E-06 | 4.0E-07 | C | NV | 3 | | | | | | - | | X |
| 822060 | Hexamethylene-1,6-diisocyanate | | | | | | | NA | | | | | | | 2B | | | | | | | | X |
| 680319 | Hexamethylphosphoramide | | | | | | | NA | | | | | | | 2B | | | | | | | | X |
| 110543 | Hexane | 19 | | X | X | X | | NA | | | | | UR | 2.0E-01 | | | | | | | | | X |
| 302012 | Hydrazine | 1 | | | | X | 13.9 | 16.1 | 13.9 | 54 | 4.9E-03 | 4.9E-05 | B2 | | 2B | | | - | | - | + | + | |
| 7647010 | Hydrochloric acid | 7 | | X | X | X | 36728.5 | 30371.2 | | 3322 | | | | 7.0E-03 | | | | | | | | | X |
| 7664393 | Hydrogen fluoride (Hydrofluoric acid) | 7 | X | X | X | X | 4278.0 | 4990.1 | 6054.5 | 527 | | | | UR | | | | | | | | | |
| 123319 | Hydroquinone | 4 | X | | X | X | 5.7 | 6.4 | 5.1 | 69 | | | | NV | 3 | | | | | | | | |
| 78591 | Isophorone | 1 | X | | | | | NA | | | | 2.7E-06 | C | NV | | | | | | | - | | X |
| 0 | Lead Compounds | 22 | | X | X | X | 812.2 | 1224.9 | 1339.7 | 923 | | | B2 | | 2B | | +- | | | -+ | - | - | |
| 58899 | Lindane (gamma-hexachlorocyclohexane) | | | | | | 0.8 | 0.4 | 0.1 | 12 | | | | NV | 2B | | | | | | | | X* |
| 108316 | Maleic anhydride | 3 | X | | X | X | 246.5 | 225.3 | 382.0 | 224 | | | | | | | | | | | | | |
| 0 | Manganese Compounds | 19 | | X | X | X | 1126.4 | 2215.4 | 1582.8 | 834 | | | D | 4.0E-04 | | B | | | | | + | + | |
| 0 | Mercury Compounds (IRIS-INORGANIC) | 11 | | X | X | X | 0.6 | 14.6 | 12.9 | 25 | | | D | 3.0E-04 | | | | | | | | | |
| 67561 | Methanol | 5 | | X | X | | 91551.6 | 99864.5 | 106892.6 | 2553 | | | | UR | | | | | | | | | X |
| 72435 | Methoxychlor | | | | | | 0.8 | 0.3 | 136.4 | 6 | | | D | NV | 3 | | | | | | | | |
| 74839 | Methyl bromide (Bromomethane) | 1 | X | | | | 1102.9 | 1289.6 | 595.8 | 43 | | | D | 5.0E-03 | 3 | | + | | + | | + | + | X |
| 74873 | Methyl chloride (Chloromethane) | 10 | X | X | X | X | 3821.9 | 4437.4 | 4693.3 | 91 | | | | UR | 3 | B | | + | + | | + | | X |
| 71556 | Methyl chloroform (1,1,1-Trichloroethane) | 3 | | X | X | X | 80699.8 | 84309.0 | 83541.5 | 3942 | | | D | UR | 3 | B | | | | | | | X |

**CURRENT DATA ON THE HAPs**
**Dec. 10,1992**

| CAS # | Chemical Name | | M Y 2 | A E 4 | C A 7 1 | T R 10 | 1990 Emis (T/yr) 2 | 1989 Emis (T/yr) 2 | 1988 Emis (T/yr) 2 | # of Emit Facil 4 | IUR per ug/m3 5 | OUR per ug/L 6 | EPA WOE 7 | RfC mg/m3 Stat 8 | IARC WOE 9 | Exp. Assoc. 10 | Gene MVV Se C 11 | Tox MVT M C 12 | Data NM B E 13 | Repr/ Dev Data 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Column Number | | | | | | | | | | | | | | | | | | | |
| | | #SC | | | | | | | | | | | | | | | | | | |
| 78933 | Methyl ethyl ketone (2-Butanone) | 20 | X | X | X | X | 60663.5 | 63815.9 | 62766.8 | 2553 | | | D | 1.0E+00 | | | | | | X |
| 60344 | Methyl hydrazine | 1 | X | | | | | | | | | | | V | | | | | | |
| 74884 | Methyl iodide (Iodomethane) | | | | | | 14.9 | 12.7 | 4.5 | 4 | | | | | 3 | | + | + | - | |
| 108101 | Methyl isobutyl ketone (Hexone) | 14 | X | X | X | X | 13655.7 | 15341.4 | 15521.4 | 1074 | | | | UR | | | | | | X |
| 624839 | Methyl isocyanate | 1 | | X | | | 7.2 | 7.5 | 5.1 | 7 | | | | NV | | | | | | X° |
| 80626 | Methyl methacrylate | 5 | X | X | X | | 1058.1 | 1571.9 | 1700.0 | 252 | | | | | 3 | | | | | X |
| 1634044 | Methyl tert butyl ether | 1 | X | | | | 1392.3 | 1495.0 | 1384.7 | 113 | | | | 5.0E-01 | | | | | | X |
| 75092 | Methylene chloride (Dichloromethane) | 9 | X | X | X | | 46248.6 | 54636.0 | 60653.4 | 1374 | 4.7E-07 | 2.1E-07 | B2 | UR | 2B | | - | - + | + | X |
| 101688 | Methylene diphenyl diisocyanate (MDI) | 1 | X | | | | 338.2 | 312.8 | 143.3 | 584 | | | | 5.0E-05 | | | | | | |
| 108394 | m-Cresol | 1 | X | | | | 3.8 | 6.3 | 9.2 | 13 | | | C | NV | | | | | | |
| 108383 | m-Xylene | 6 | X | | X | | 601.1 | 583.7 | 1012.1 | 58 | | | | NV | | | - | | - | X |
| 121697 | N,N-Dimethyl aniline | 1 | X | | | | 25.4 | 45.9 | 49.5 | 22 | | | | | | | | | | |
| 91203 | Naphthalene | 2 | X | X | | | 1853.1 | 1656.9 | 1932.0 | 410 | 4.2E-06 | | C | | | | - | | - | |
| 0 | Nickel Compounds (subsulfide) | 16 | | X | X | X | 132.8 | 466.5 | 284.1 | 734 | 4.8E-04 | | A | UR | 1 | B | + - | -+ + | + - | |
| 98953 | Nitrobenzene | 1 | X | | | | 33.1 | 19.4 | 19.6 | 18 | | | D | UR | | | - | | - | X |
| 62759 | N-Nitrosodimethylamine | 1 | | | X | | | NA | | | 1.4E-02 | 1.4E-03 | B2 | | 2A | | | - + | + + | |
| 59892 | N-Nitrosomorpholine | | | | | | | NA | | | | | | | 2B | | | | | |
| 684935 | N-Nitroso-N-methylurea | | | | | | | NA | | | | | B2 | | | | | | | |
| 90040 | o-Anisidine | 1 | X | | | | 0.9 | 1.0 | 1.1 | 7 | | | | NV | 2B | | | | | |
| 95487 | o-Cresol | 1 | X | | | | 19.6 | 29.8 | 44.5 | 30 | | | C | NV | | | | | | |
| 95534 | o-Toluidine | | | | | | 3.7 | 12.8 | 23.5 | 20 | | | | | 2B | | - | +- +- | - - | |
| 95476 | o-Xylene | 23 | X | X | X | X | 952.3 | 899.6 | 979.6 | 83 | | | | NV | | | - | | - | X |
| 56382 | Parathion | | | | | | 0.2 | 0.8 | 1.6 | 10 | | | C | | 3 | | | | | |
| 82688 | Pentachloronitrobenzene (Quintobenzene) | | | | | | 0.1 | 1.0 | 0.5 | 5 | | | UR | | 3 | | | | | |
| 87865 | Pentachlorophenol | 4 | X | | X | X | 11.6 | 5.6 | 7.1 | 48 | | 3.0E-06 | B2 | UR | 2B | | | - + | - | |
| 108952 | Phenol | 12 | X | X | X | X | 3827.4 | 5264.0 | 5083.3 | 678 | | | D | NV | | B | - | + | v | |
| 75445 | Phosgene | 2 | X | | X | | 2.4 | 4.1 | 10.8 | 34 | | | | NV | | | | | | |
| 7803512 | Phosphine | | | | | | | NA | | | | | D | | | | | | | |
| 7723140 | Phosphorus | 5 | | X | X | X | 12.1 | 30.1 | 9.6 | 70 | | | D | | | | | | | |
| 85449 | Phthalic anhydride | | X | | X | X | 343.7 | 325.0 | 273.6 | 185 | | | | | | | | | | |
| 1336363 | Polychlorinated biphenyls (PCB's) | | | | | | 0.0 | NA | 0.1 | 39 | | 2.2E-04 | B2 | | 2A | | - - | - | - | X |
| 0 | Polycyclic Organic Matter | 12 | | X | X | X | | NA | | | | | UR | | | B | | | | |
| 123386 | Propionaldehyde | 4 | X | | | X | 494.5 | 453.8 | 523.1 | 18 | | | | | | | | | | |

**CURRENT DATA ON THE HAPs**
**Dec. 10,1992**

| CAS # | Chemical Name | M Y 1 | A K 4 | C A 7 | T R 10 | 1990 Emis (T/yr) | 1989 Emis (T/yr) | 1984 Emis (T/yr) | # of Emit Facil | IUR per ug/m3 | OUR per ug/L | EPA WOE | HEI mg/m3 Stat | LARC WOE | Reg. Assess. | Gene MVV Se | Gene MVV G | Tox MVT M | Tox MVT G | Data NM S | Data NM E | Repro/ Dev Data |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Column Number | | | 1 | | 2 | 3 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | | 12 | | 13 | | 14 |
| | 880 | | | | | | | | | | | | | | | | | | | | | |
| 114261 | Propoxur (Baygon) | | | | | 0.1 | 0.3 | 0.1 | 7 | | | UR | | | | | | | | | | |
| 78875 | Propylene dichloride (1,2-Dichloropropane) | 1 | X | | | 315.3 | 616.7 | 662.1 | 12 | | | UR | 6.0E-03 | 3 | | | | | | | | X |
| 75569 | Propylene oxide | 16 | X | X | X | 660.0 | 897.1 | 1462.8 | 128 | 3.7E-06 | 6.8E-06 | B2 | 3.0E-02 | 2A | | + | - | + | + | + | + | |
| 106445 | p-Cresol | 1 | X | | | 119.5 | 127.6 | 320.4 | 16 | | | C | NV | | | | | | | | | |
| 106503 | p-Phenylenediamine | 1 | X | | | 0.4 | 2.0 | 56.9 | 11 | | | | | 3 | | | | | | | | |
| 106423 | p-Xylene | 3 | X | | X | 2969.3 | 2360.0 | 3153.0 | 43 | | | | NV | | | - | | | | - | | X |
| 91225 | Quinoline | | | | | 13.8 | 31.8 | 24.7 | 26 | | | | NV | | | | | | | | | |
| 106514 | Quinone (1,4-benzoquinone) | 1 | X | | | 0.8 | 0.9 | 5.7 | 4 | | | | NV | | | | | | | | | |
| 0 | Radionuclides (including radon) | | | | | | | | | | | | | | A | | | | | | | |
| 0 | Selenium Compounds | 15 | | X | X | 15.3 | 16.7 | 14.8 | 46 | | | D | | 3 | | | | | | | | |
| 100425 | Styrene | 15 | X | X | X | 15838.3 | 16650.9 | 17386.3 | 1380 | | | UR | V | 2B | | +- | | + | | + | | X* |
| 96093 | Styrene oxide | | | | | 1.2 | 0.4 | 1.2 | 5 | | | | | 2A | | - | - | + | + | + | + | X |
| 127184 | Tetrachloroethylene (Perchloroethylene) | 11 | X | X | X | 10622.5 | 12752.4 | 15794.3 | 620 | | | B2 | | 2B | B | | - | + | | - | - | X |
| 7550450 | Titanium tetrachloride | | | | | 27.2 | 28.6 | 39.3 | 39 | | | | | | | | | | | | | |
| 108883 | Toluene | 39 | X | X | X | 116912.8 | 127718.9 | 135611.9 | 4006 | | | D | 4.0E-01 | | B | + | | | - | - | | X* |
| 8001352 | Toxaphene (chlorinated camphene) | | | | | | NA | | | 3.2E-04 | 3.2E-05 | B2 | | 2B | | | - | | | + | | |
| 79016 | Trichloroethylene | 8 | X | X | X | 16949.0 | 22162.8 | 24092.3 | 766 | 1.7E-06 | 3.2E-07 | B2 | UR | 3 | B | + | - | +- | +- | + | + | X |
| 121448 | Triethylamine | 1 | X | | | | NA | | | | | | 7.0E-03 | | | | | | | | | |
| 1582098 | Trifluralin | | | | | 7.8 | 2.0 | 1.6 | 22 | | 2.2E-07 | G | | | | | | | | | | |
| 108054 | Vinyl acetate | 5 | X | | X | 2778.4 | 2699.1 | 2869.6 | 152 | | | UR | 2.0E-01 | 3 | | | | | + | - | | X |
| 593602 | Vinyl bromide (bromoethene) | | | | | 5.1 | 0.4 | 2.5 | 2 | | | | | 2A | | | | | | + | | |
| 75014 | Vinyl chloride | 4 | X | | X | 567.9 | 634.4 | 667.2 | 52 | 8.4E-05 | 5.4E-05 | A | | 1 | A | + | - | + | | + | + | X* |
| 75354 | Vinylidene chloride (1,1-Dichloroethylene) | 3 | X | | X | 151.8 | 110.3 | 149.7 | 21 | 5.0E-05 | 1.7E-05 | C | UR | 3 | B | - | - | - | - | + | + | X |
| 1330207 | Xylenes (isomers and mixture) | 23 | X | X | X | 69968.4 | 73743.4 | 71332.8 | 3547 | | | D | NV | | | - | | | - | - | - | X |

IV. Question 4. What does EPA consider to be the prioritization of the information gathering needs? What criteria would EPA use for determining this prioritization?

## IV.A Introduction

Existing data on effects and exposure to the hazardous air pollutants (HAPs) listed under Section 112 have supported a variety of decisions under Title III of the Clean Air Act (CAA). Rules that use these data and additional data collected in a timely fashion will continue to be issued on CAA schedules that extend to the year 2010. Future information gathering on the HAPs will support residual risk decisions, biennial Great Waters reports, urban air toxics reports, and other continuous activities required to administer Section 112 provisions. Interest in the HAPs exists beyond the CAA. Other EPA-administered programs and programs of other agencies address many of the same chemicals and mixtures. Therefore, whatever data are gathered will be gathered with an eye to serving needs beyond Section 112.

The process of prioritizing data collection activities must consider many factors. Decisions for gathering information will have both science and management components. Important considerations include: the types of information needed to make a statutory finding, the current state-of-the-science, priorities given other EPA work, budget constraints, and statutory deadlines. The EPA has not, as yet, made decisions about the extent, mechanism, or timing of data gathering activities. The information presented below generally describes EPA's initial thoughts regarding the gathering of information needed to effectively implement Title III of the CAA.

Under Title IX of the CAA, EPA and other agencies will be looking generally at the research needs for all of the HAPs. This Title provides a forum for planning research to advance the state of the art beyond standard testing. The plans for carrying out this Title are currently being formulated as the Title was added after the FY91 appropriation process was completed.

Overall, the goals by which the priorities and needs can be balanced may be stated as:

- ensure that the data collected meet the requirements of the statutory finding(s) that must be made
- ensure that the data are collected in a timely fashion
- ensure efficient use of resources, given the parallel data gathering efforts of others
- ensure that adequate resources are invested in HAPs that are emitted in significant volumes
- avoid enriching an already rich data base of one HAP at the expense of another HAP of importance

## IV.B Criteria for Effects Data-Gathering Plan

The major focus in planning for health and environmental effects data collection activities is to ensure that adequate data are available to conduct the residual risk determinations that will be made under Section 112(f). In order to obtain the data necessary to support these decisions required later in the decade, EPA must begin collection efforts immediately. The Agency anticipates that activities will begin with a ranking of HAPs that takes several factors into account. These factors include:

- promulgation dates of control technology standards
- estimation of the extent to which a particular HAP will contribute to risks resulting from combined HAP emissions from sources in a source category (using effects and exposure data available now)
- importance of a HAP to the Great Waters or Urban Area Source programs
- overlapping priority/interest of other EPA programs or governmental agencies (e.g. timing of ongoing Agency for Toxic Substances and Disease Registry or National Toxicology Program activities)

Decisions on the extent and type of data to be gathered on potential adverse effects associated with exposure to a HAP will also require a balancing of several factors including incorporation of professional judgment on the likelihood that additional data may significantly alter current opinions on the toxicity of a specific HAP. Critical elements will include:

- the richness of the current data base
- the need for data to enable route-to-route extrapolation of existing toxicity data
- the need to expand a data set on an already identified endpoint in order to improve dose-response characterization
- the need to extend the scope of data to cover endpoints other than those previously identified
- the need for research beyond standard test protocols to understand biological fate and transformation or mechanism of action

## IV.C Options for Scope of Effects Data-gathering

While the alternatives have not been exhaustively explored, and substantial work remains to be done, there are three general options that are being considered. These options are:

1. Broad Scope. This approach would use staged testing for a large number of HAPs, screening a range of endpoints and proceeding to full endpoint tests as the screening assays indicate.

2. Medium Scope. This option would focus screening tests on those HAPs with the most significant emissions. Testing strategies would be more robust and address critical endpoints (carcinogenicity and developmental toxicity, at a minimum). Other HAPs with significant emissions would be considered under the narrow scope testing identified below.

3. Narrow Scope. Under this alternative, testing would focus on complimenting and making more useable existing data bases. For example, HAPs with significant emissions may be studied to "convert" oral to inhalation data or to elucidate dose-response relationships. This narrow scope testing could include: pharmacokinetics studies, a 90 day subchronic inhalation study, or a repeat of a previous study on an endpoint to better define the dose-response relationship.

## IV.D Mechanisms for Obtaining Effects Data

There are a variety of mechanisms that may be accessed for collecting effects data, all of which will likely be employed. Major data gathering efforts are underway that will complement data collected specifically for Section 112 use. For example, the Superfund Amendments and Reauthorization Act of 1986 (SARA) requires the Agency for Toxic Substances and Disease Registry (ATSDR) to prepare toxicity profiles for over 200 pollutants. These profiles identify data gaps and efforts will be put forth to fill these gaps. Of the pollutants studied by ATSDR, 76 are HAPs. A second example is efforts being undertaken by the European communities. They are interested in generating data for a list of chemicals that overlaps the HAPs list. In addition, the National Toxicology Program (NTP) is working with EPA to identify testing and research the NTP can undertake for several HAPs. The EPA's Health and Environmental Research Laboratory (HERL) has ongoing research that addresses several HAPs, as well as urban toxics issues. This laboratory also conducts fundamental research on pharmacokinetics applicable to the HAPs. Additional EPA laboratories are conducting research on environmental fate, ecological effects, etc. Another alternative for collecting data is to access the regulatory test program under the Toxic Substances Control Act (TSCA) to require that industry conduct testing. Finally, the CAA Title IX research program will be pursued for research on HAPs. Making these overlapping efforts work together will be part of any data gathering EPA does on the HAPs.

## IV.E Improving Data Bases for Estimating Exposure to HAPs

In addition to developing quantitative relationships between HAP concentrations and health or environmental effects, it is critical that the EPA pursue parallel efforts to support accurate characterization of the levels of exposure associated with sources of the HAPs. In the past, efforts to obtain sufficient information to accurately characterize HAP exposure levels in the vicinity of an industrial source

have focused on one pollutant at a time. These efforts have been severely limited by lack of information on the source(s) being evaluated. In lieu of site-specific data for exposure characterization, EPA has settled for "model plant" types of analyses, which rely on only a sampling of data from one type of source and extrapolate exposure estimates to the rest of the source population. These analyses by nature must be very conservative, and therefore tend to overestimate ambient exposure levels due to any one type of industrial source. As a result, these analyses are often criticized by industry as being "overly conservative".

It is clear that the CAA mandate for residual risk analyses (after the implementation of MACT) would require that such analyses be based on site-specific data rather than "model plant" scenarios. These analyses must therefore require more site-specific data than are currently available. In addition, the analyses will differ from past analyses in that they will be directed at assessing the exposure to multiple pollutants being emitted from a source in a particular source category. The EPA must begin now to develop the tools and process for obtaining the necessary data to perform residual risk analyses. While such efforts may build on past efforts, there are several new and challenging aspects that must be addressed, including:

1) Emission levels of each of the HAPs from each source within a source category must be obtained. Since EPA-approved measurement methods are not available for all HAPs, this will entail research and development efforts for both measurement methods and site-specific emission estimation techniques. It is hoped that cooperative efforts can be undertaken with industry to expand the publicly-available expertise in this area.

2) Data are to be obtained on a source category-by-source category basis. Since most currently available data bases are on a pollutant-by-pollutant basis, most of the current data will be inadequate for this purpose.

3) Exact stack, vent, and fugitive emission locations as well as fenceline locations for each facility are crucial for reducing the uncertainty of exposure assessments. Very little data are available in this regard, and it is unclear whether most industries will be willing to provide such data.

4) Development of guidelines is needed to explore the use of monitoring data or other more direct measures of exposure in assessing exposures resulting from emissions of HAPS. Specifically, the use of these data to complement modeling analyses.

5) Development of a user-friendly, easy-access, centralized data base and retrieval system (such as an electronic bulletin board system) may be desirable to provide a convenient vehicle for obtaining the necessary data. Industry input and cooperation in such development would be crucial to its success. Making

sure that industry realizes that, without the necessary data, EPA efforts to assess exposure will be "conservative", may provide the needed cooperation of industry. Development of a data base system that is easy to use will substantially reduce the burden on industry as well as reduce the paperwork that would otherwise be necessary for such an information request.

6) Efforts to check and assure the quality of the data obtained for exposure assessments may prove to be a large part of the data gathering process.

7) Efforts to appropriately include population mobility and microenvironment exposures into the overall exposure assessment process have already begun. Sensitivity studies are needed, however, to determine the extent to which such factors can affect the overall exposure and risk assessment results.

8) Inclusion of short-term exposure quantification is important for many HAPs. Some modeling techniques are already available to address this quantification, but data on short-term emissions variability are generally lacking. The extent to which such information becomes available will dictate the extent to which EPA can incorporate such variability in exposure assessments.

9) Concentration measurements to assist in the validation of human exposure modeling results are generally lacking for most HAPs. While validation of air dispersion models in the field has been done, indoor/outdoor partitioning and multiple route exposures have not received the same level of validation efforts. This is an area where more data would be helpful.

EPA welcomes comments and suggestions from the Committee on the plans for improving the accuracy of exposure and risk assessments required to implement the CAA. Of specific interest are the recommendations of the panel for prioritizing the vast amount of work that is required to fill the existing data gaps.

V. Question 5: What does EPA consider to be some of the critical management aspects of risk assessment decision-making that may not be apparent to an outside observer?

The current regulatory process places a number of challenging demands on the risk manager. Depending on the nature of the regulation and the legislative authority, he or she must try to assimilate a variety of analyses -- legal, economic, social and scientific -- of which risk characterization is only one part. Because of this diversity, the risk manager must relay on the products of experts in a range of disciplines.

In making risk management decisions, there are a number of considerations and factors to be weighed that may not be apparent to outside observers. Some of the factors influencing these decisions are described below.

1. In dealing with scientific issues, the risk manager is typically a generalist with no particular expertise in the area of risk assessment. This places particular requirements on the risk assessment process. Thus, the products of the risk assessment process must be designed to aid these individuals in decision-making. Risk managers are often frustrated by complex discussions of scientific uncertainties (mechanism of action, uncertainty in extrapolation, etc.). Rather they tend to desire bottom-line characterizations of the likelihood and magnitude of potential problems. In many respects, the popularity of the current cancer classification system lies in its ability to characterize the overall weight of evidence by readily- comprehended categories (e.g., known, probable, possible carcinogen) and the presentation of a measure of carcinogenic potency.

The Agency has increased the emphasis placed on the risk characterization component of risk assessment, and is moving toward a more comprehensive examination of the assumptions and uncertainties in risk assessment. The fact remains, however, that communication of the critical elements of a risk assessment to risk managers remains a challenge.

2. Consistency is important. This does not mean that all risk assessments should look the same. But it is important that a consistent terminology be adopted, even if the terminology draws controversy, and that the risk managers understand and can communicate that understanding. Decision-makers build on previous decisions and examples to put current issues in context. If formats or meanings differ from case to case, this process becomes difficult, if not impossible.

3. Risk managers do not expect perfect information. Critics of risk assessment's imperfections must recognize that public policy is often a blunt instrument rather than a surgeon's scalpel. Decisions are often based on broad bands of uncertainty within which even differences of several fold may not affect the decision.

It is important for both risk managers and critics of risk assessment to avoid pursuing the ideal risk assessment. These individuals must bear in mind the limits of the real world. These limits include time, money, and the state of scientific knowledge.

4. Statutory mandates may place constraints on the development and use of risk characterization data that are not consistent with our understanding of the underlying science. The establishment of risk targets (or bright lines) such as $10^{-6}$, for example, have been criticized as not allowing the consideration of weight-of-evidence in decision making. Another example is the requirement that the Agency consider the risk to the "person most exposed" to emissions from an air toxics source. Thus, the statutory framework constrains full consideration of the distribution of risk across the exposed population.

5. Statutes or court action often mandate regulation at a specific time, effectively mandating decision-making based upon available data. This is exacerbated by the fact that the development of robust health and safety data (e.g., well-conducted animal bioassays, epidemiological, or exposure studies) are both resource- and time-intensive.

6. The risk management process is often the focus of considerable outside attention and controversy. This is particularly true where the impacts of decisions are costly, or where they adversely affect well-organized groups. On these circumstances, there is a natural tendency to continue the process of data development and analysis, rather than to make decisions in an atmosphere of uncertainty. While such an environment can cause delay, it can also have the effect of encouraging more rigorous examination of data and careful consideration of options.

7. Persistent requests for information and more studies lead to paralysis by analysis and the waste of limited resources. The risk of inaction is often forgotten. Additional information needs must be balanced against the need to take timely action where it is warranted. This is particularly true in the risk assessment process, where the limitations of the current state of the science often prevent definitive answers, and can encourage continual additional data development. Reviewers of Agency risk assessments must consider the reasonable resource constraints under which the Agency operates.